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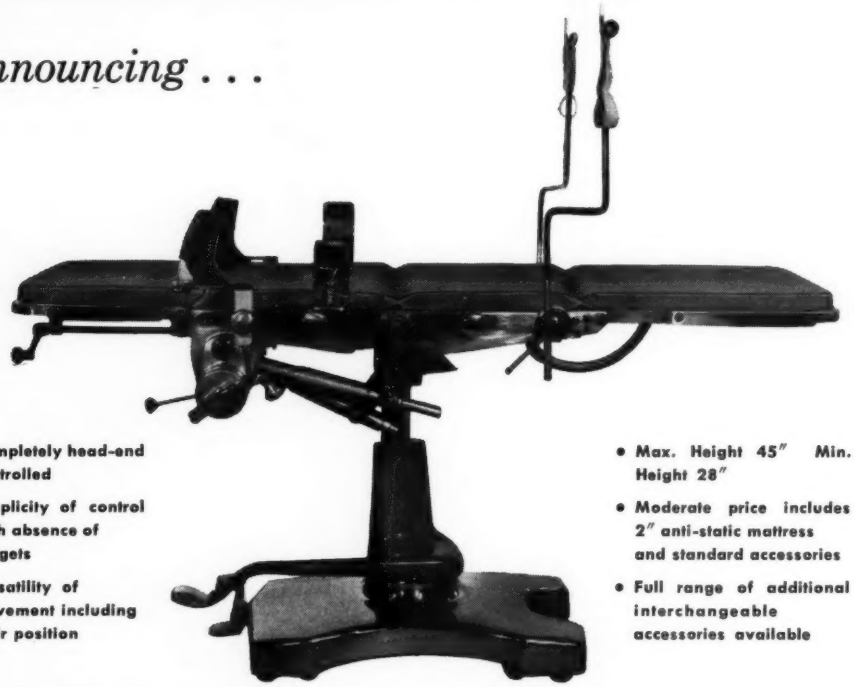

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# The Manitoba Medical Review

Vol. 37 AUGUST - SEPTEMBER, 1957 No. 7

## Psychiatry

### Listening to Patients

W. Donald Ross, M.D., B.Sc. (Med.) (Man.), F.R.C.P. (C)

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The daughter of a psychiatrist friend of mine was being teased by another doctor who said, "Your daddy doesn't **do** anything for patients; he just talks to them." With a composure as unusual among psychiatrists' children as is good behaviour among ministers' children, she replied: "He doesn't talk to them; he just listens to them." This, of course, was a profound truth. I hope to explain to you in this hour that the point of the story was **not** that psychiatrists don't do anything for patients, but rather that skillful listening, whether by a psychiatrist or any other doctor, is doing a great deal for patients. We might modify this girl's phrase from "just listens" to "listens justly" or judiciously, to indicate that listening with good judgment is the core of psychiatric skill. It can be the core of any physician's skill at helping people with the emotional problems which either brought them to the doctor without significant organic disease, which contributed through psychosomatic mechanisms to the organic disease, or which are preventing the patient from making the best use of the physician's or surgeon's attempts to do more than listening.

Listening with good judgment means listening with a purpose. The two purposes for listening to patients which I shall discuss are: listening for diagnosis, and listening for treatment.

Listening for diagnosis expresses the essentials of medical interviewing, a term which can be used to characterize the technique of a skilled clinician in contrast with the rigid "history taking" of a second year medical student. I am sure you have all modified your "history taking" technique a great deal since your days as a medical undergraduate. Perhaps some of the principles of interviewing, which revolve around good listening, can help you to modify your medical interviewing even further, with advantage to yourself and your patients from more complete diagnosis, that is, more complete understanding of the factors contributing to and complicating your patient's illnesses.

One experiment at a hospital connected with Harvard University illustrated the fact that training in rigid, question and answer, history taking had at least temporarily hampered the students' original natural aptitudes for interviewing. In this experiment the same patients on the medical wards were interviewed by senior medical students and senior arts students. The shocking finding was that the senior arts students elicited more information from the patients concerning emotional and social problems relevant to their illnesses than did the medical students! Of course the arts students did not know how to use the information. Nevertheless they appeared to have a skill which the medical students presumably had lost. One could not say that the particular species, homo studens medicinae, never had the required attributes. Those who enter the study of medicine are particularly endowed with the attitude of benign curiosity about the inner workings, both physical and mental, of other people, which is necessary to be a good interviewer. The difficulty must lie in adhering too rigidly to "history taking" which may force the patient's utterances into a Procrustean bed which leaves little room for individual emotional and social experiences of the patient.

However, we do like some "rules" to follow as well as a general purpose. So I shall give you some rules for interviewing to help you in modifying your "medical interviewing" further away from the stultifying quiz technique of your medical student days.

**Rules for Interviewing**

1. Observe privacy and confidentiality.
2. Give your full attention to the person being interviewed.
3. Indicate interest in the problem of which he is aware.
4. **Listen; don't talk.**
5. Never argue; never give advice prematurely.
6. Listen to a) what he wants to say,
   b) what he does not want to say,
   and c) what he cannot say without help.
7. As you listen, plot out the pattern which is being set before you.
8. Reserve helping him to say more until you have a substantial sample of his spontaneous pattern.

These "rules," of course, are to be applied flexibly, and they will be discussed in further detail with regard to exceptions and qualifications, but it cannot be overemphasized that physicians habitually err on the side of asking too many direct questions too soon, and of giving advice before enough evidence is in.

1. **Observe Privacy and Confidentiality**

This rule may seem obvious but one encounters first rate physicians who have been seeing patients in an open dispensary, with other people milling around, and who were surprised that they were

Lecture in Annual Refresher Course, University of Manitoba Faculty of Medicine, April 10, 1957.

not able to elicit the personal problems which they inferred to be present behind the symptoms.

**2. Give Your Full Attention to the Person Being Interviewed.**

Your genuine interest in the person you are interviewing and your benign curiosity about his problems will be evident to him if you do not allow yourself to be distracted or preoccupied with other matters To have the full interest of someone, especially of an "expert," is so encouraging that most people with problems, unless they are unusually defensive, will open up with little need for priming.

**3. Indicate Interest in the Problem of which He is Aware**

If he has asked to talk to you, or if he is presenting symptoms for your medical advice, he will talk most readily at first about what appears to be the main problem to him. Even if you are eager to get on to learning more about him, medically or psychologically, don't cut him short on what he is wanting to say to you.

**4. Listen; Don't Talk**

If you interrupt with questions or comments you will set up a pattern in which the person you are interviewing leaves it to you to drag out all his feelings and all the facts. If you don't interrupt you will be able to fill in the gaps by questions later. If you cut him off during his initial start you will have a hard time getting back to spontaneous productions from him. If he is hesitant, you may have to encourage him by comments indicating your interest, or by phrases such as "Tell me more about it," "Why?" "For instance?" or "For example?" or by repeating something he has said. If he is motivated to confide a problem to you, and if he is aware that the more you know about him, the more you can help him, then you can even accept lapses into silence without feeling you have to break the silence, and you may find that something particularly significant was behind the silence. A busy physician often feels that he must get on with collecting all the facts he can from a patient in as short a time as possible. But this often results in waste of time in the long run since it leaves out the possibility of learning from the patient the particular feelings and facts which contain the key to the problem.

Starting from his symptoms or problem, he will digress to other personal and social information and your picture of him can be rounded out much more efficiently by encouraging a line in his verbalizations than it could be by taking "family history" and "personal history" in chronological order.

**5. Never Argue; Never Give Advice Prematurely**

The attitude of tolerance is so important for the obtaining of an honest report of feelings and of facts, any indication of disagreement or of advice, or of judgmental attitudes, is bound to result in the suppression of the very material which the doctor needs in order to understand the person he is interviewing.

**6. LISTEN TO A) WHAT HE WANTS TO SAY,**
**B) WHAT HE DOES NOT WANT TO SAY,**
**C) WHAT HE CANNOT SAY WITHOUT HELP.**

**7. AS YOU LISTEN, PLOT OUT THE PATTERN WHICH IS BEING SET BEFORE YOU.**

These two rules can be considered best together since one's understanding of what the person wants to say, and does not want to say, or cannot say without help, grows as one observes the pattern being followed and considers it in the entire psychological context. One keeps in mind not only what the person is saying, but asks oneself the question, "Why is he saying this?" What he wants to say contains much of his defenses. What he does not want to say is usually because of anxiety about this material. To test one's hypotheses and to correct them as one goes along one can make comments to the person which summarize what he has wanted to say, or hint at what he has not wanted to say, and if this is done cautiously, without adding or distorting, it can be helpful in encouraging him to say more of what he does not want to say.

For example, a coal miner being interviewed for the assessment of emotional factors in his symptom of shortness of breath, was noted to digress from physical symptoms to complaints about the dustiness of the mines and to make a passing reference to another miner who had been killed in a rock fall. Then he skirted away from this subject to symptoms again. Later it was indicated to him that he had mentioned this death only briefly but that perhaps it had bothered him a good deal. He admitted that he didn't like to talk about it but that he had worried considerably about it. He began to open up on a change in his own feelings about the hazards of mining following upon his friend's death, and how he had worked all the harder on extra shifts to save up more money for his family in case something happened to him.

This aspect of interviewing, of feeling with the subject, and inferring the layers of defense, anxiety, and basic potentialities, improves with practice. The method of inferring what is behind what a person is saying can be likened to the astronomical method of measuring the distance to a star by "triangulation" from different observation points. If one observes that a person has talked about two subjects consecutively which are not obviously related and yet which would be related if one infers a third subject linking the other two, one may be able to conjecture correctly what he has in "the back of his mind."

8. **Reserve Helping Him to Say More Until You Have a Substantial Sample of His Spontaneous Pattern.**

Your "triangulations" as to what is behind what he is saying must not be brought to his notice too soon or he may fail to give you enough of what he wants to say and what he can say without your help. But if you have enough from him to be sure of what is behind his words, something which he has not verbalized but of which he may be aware, you may be able to verbalize it for him and thus give him such a feeling of being understood, that he will open up all the more. Practice will enable you to learn when this can be done. It works when the comments are along the lines that people with similar problems have tended to get to next. It does not work if the comments are about feelings or facts against which the individual is particularly defensive.

The filling in of gaps in a detailed medical and personal history comes in the category of helping the patient to say more after you have given him plenty of opportunity, through direct statements and digressions, to tell you about himself. How complete a history you will want will depend on the nature of the problem and on your medical judgment. Except for such questions as fit in naturally with the flow of the patient's own story, one can actually postpone direct medical questions until during and after physical examination.

The **physical examination** is itself a method of helping the patient to say more. Particularly significant material may be proffered by the patient during and following the physical examination.

Following these rules of interviewing rather than a "history plan" will elicit from most patients surprisingly complete information toward a diagnosis, which can always be supplemented by further questioning, whereas a barrage of questions directed at a patient before he needs help to say more may shut him up so that one has lost the opportunity to get a sample of his own stream of words, thoughts and feelings.

I shall not depart from the listening theme to the extent of going over the categories of items for a medical history on which there may be some need to give the patient help to say more. However I might mention that there is an order of topics in personal history which corresponds with relative degrees of ease in talking. If one has to do some priming to help the patient to say more, this priming will succeed best if it is done in the following order. 1. health history, 2. occupational history, 3. religious background and history, 4. family history, 5. childhood history, 6. sex history and only finally 7. recent personal events not previously divulged. The latter may contain the most significant keys to the precipitation of the presenting problem.

Listening for diagnosis means listening until you understand, not only the clinical diagnosis, but how this patient came to have this illness or this personal reaction at this time. Only if one has listened to this extent can one avoid the frequent errors of omission and commission in diagnosis—the errors of labelling something "neurotic" when it is "organic," or, vice versa, or of failing to realize that both "emotional" and "organic" aspects must be treated.

This brings us to listening for treatment.

The type of interviewing already outlined is not only a diagnostic technique. It is a therapeutic technique, especially if repeated in judicious doses at intervals. It is the core of psychotherapy. All physicians practice psychotherapy. They may not call it this. They may do it well or they may do it badly. But whether they recognize what they are doing or not, the relationship which develops between doctor and patient in any medical or surgical situation has psychological effects on the patient for better or for worse.

Psychotherapy is listening plus saying things which help the patient to become less anxious or to make progress in solving the conflicts which gave rise to the anxiety. We cannot pursue in detail in this hour the more than listening which can be done by general physicians as psychotherapists. However we can emphasize that listening is "trumps," and, when in doubt, play trump. Many doctors fail to realize the full emotional value to patients of having someone important who will listen to them.

A story is told about Dr. Helene Deutsch, a psychoanalyst in Boston, during a period when she was living in a hotel after she first came to the United States from Vienna. A room clerk, having heard of her skill, sought one interview with her to talk over some problems. Years later she met him and he told her that she had "helped him to clear up his difficulties completely." The punch line of the story is this: that at the time of the interview she had had great difficulty in understanding his English! Her comment on the story is that, "It certainly proves that a good therapeutic result is not always due to a good understanding."

Since I have hinted at further information which could not be covered in this hour I want to suggest some additional reading. I commend to you particularly a handy pocket book by another former Manitoban now in Ohio—Brian Bird; its title is "Talking with Patients," published by Lippincott, 1955. Further references as well as further details are presented also in Chapters 3 and 6 of a book by myself "Practical Psychiatry for Industrial Physicians" published by Charles Thomas, 1956.

# Medicine

## Nutritional Disorders of the Nervous System

### Part II

Maurice Victor, M.D.

**Korsakoff's Psychosis**— Between the years 1887 and 1889, the Russian psychiatrist, S. S. Korsakoff, in a series of three papers, delineated the characteristic mental disorder which now bears his name. In his first article he drew attention to the psychic symptoms that so frequently accompany alcoholic neuropathy, and proposed that the psychic disturbance and the neuropathy represent "two facets of the same disease" . . . "The pathogenic cause provoking multiple neuritis may affect several parts of the nervous system, central as well as peripheral, and acording to where this cause is localized there will be symptoms either of neuritis or of the brain." Because of this close associationship he proposed the name "psychosis associated with polyneuritis," or "polyneuritic psychosis." In the second article (1889), Korsakoff brought out a fact which is still not fully appreciated today— that neuropathy need not accompany the characteristic mental syndrome. In his own words: "At times . . . the symptoms of multiple neuritis may be so slight that the whole disease manifests itself exclusively by psychic symptoms." In this article he stated his views on the etiology of the mental illness: "I call this form toxemic, because I consider the disturbance of brain in this form closely connected with some form of toxemia. It is connected with the presence in the blood of some poisonous substances, some toxins." He suggested that the name be changed to "cerebropathia psychica toxaemica." The third article in this series represents the summation of Korsakoff's ripened clinical experience. An excerpt is reproduced below.

"Together with the confusion, nearly always a profound disorder of memory is observed, although at times the disorder of memory occurs in pure form. In such instances the disorder of memory manifests itself in an extraordinary peculiar amnesia, in which the memory of recent events, those which just happened, is chiefly disturbed, whereas the remote past is remembered fairly well. Mostly the amnesia of this particular type develops after prodromal agitation with confusion. This excitement may last several days and then the patient again becomes calm and his consciousness clears; he appears to be in better possession of his faculties; he receives information correctly, and yet his memory remains deeply affected. This reveals itself primarily in that the patient constantly asks the same questions and repeats the same stories. At first, during conversation with such a patient it is difficult to note the presence of psychic disorder; the patient gives the impression of a person in complete possession of his faculties; he reasons about everything perfectly well, draws correct deductions from given premises, makes witty remarks, plays chess or a game of cards, in a word, comports himself as a mentally sound person. Only after a long conversation with the patient, one may note that at times he utterly confuses events and that he remembers absolutely nothing of what goes on around him: he does not remember whether he had his dinner, whether he was out of bed. On occasion the patient forgets what happened to him just an instant ago: you came in, conversed with him, and stepped out for one minute; then you come in again and the patient has absolutely no recollection that you had already been with him . . . In conversation they may repeat the same thing 20 times, remaining wholly unaware that they are repeating the same thing in absolutely stereotyped expressions. It often happens that the patient is unable to remember . . . his attending physician or nurse, so that each time he sees them, even though seeing them constantly, he swears that he sees them for the first time.

With all this, the remarkable fact is that, forgetting all events which have just occurred, the patients usually remember quite accurately the past events which occurred long before the illness. What is forgotten usually proves to be everything that happened during the illness and a short time before the beginning of the illness. Such is the case in the more typical instances of the disease; in others, even the memory of remote events may also be disturbed.

. . . "The amnestic manifestations vary. In milder degrees for example, there may be no complete abrogation of the memory of recent events, only the facts are remembered vaguely, unclearly. In some cases the facts themselves are remembered, but not the time when they occurred . . . At times . . . they may believe themselves to be in the setting (or circumstances) in which they were some 30 years ago, and mistake persons who are around them now for people who were around them at that time but who are now perhaps even dead . . . The memory often improves . . . so that if the disease progresses toward improvement the amnesia diminishes and may entirely disappear . . . In regard to the confusion, it must be noted that in this form of amnesia a slight degree of confusion is frequently present . . . When asked to tell how he has been spending his time, the patient would very frequently relate a story altogether different from that which actually occurred, for example, he would tell that yesterday he took a ride in town, whereas, in fact, he has been in bed for two months, or he would tell of conversations which have never

*From the Neurology Service, The Massachusetts General Hospital, Boston, Mass. This article is an enlarged version of a lecture to the section on Internal Medicine of the Manitoba Medical Association, October, 1956.

occurred, and so forth. On occasion, such patients invent some fiction and constantly repeat it, so that a peculiar delirium develops, rooted in false recollections (pseudo-reminiscences)."

Korsakoff's description remains remarkably fresh to the present day, although neither of the names he suggested has survived. The term toxic was soon dropped, since Jolly pointed out that the characteristic mental disturbance could be found under circumstances in which a toxin could not possibly be implicated, such as following head injury. He proposed the term Korsakoff's syndrome, since we are not dealing with a disease sui generis. Bonhoeffer suggested further that the term Korsakoff's phychosis be reserved for the purely alcoholic forms of the disease, while the forms unconnected with alcoholism be called Korsakoff's syndrome. Some authors prefer the term amnestic confabulatory syndrome, or simply amnestic psychosis where mental symptoms are present without neuritic ones. The terms Korsakoff's psychosis, Korsakoff's syndrome and amnestic-confabulatory syndrome are now used interchangeably, despite the attempts to give them specific connotations.

The term Korsakoff's psychosis is generally characterized as a state of "memory defect with confabulation." This feature alone fails to adequately describe this psychosis. It was apparent from Korsakoff's writings that his patients displayed a much wider range of symptoms, including those of delirium and what he termed "irritable weakness" (anxiety, fear and depression). Our patients with Korsakoff's psychosis showed, in addition, a wide range of intellectual impairment. If one subjects these patients to formal psychological testing, they are found defective in concentration, verbal and visual abstraction, in visual-motor coordination, and in learning ability. Although such a pattern is probably not specific for Korsakoff's psychosis, it is worth emphasizing that there are in this disease a number of abnormalities in cognitive function apart from those of memory defect. Nevertheless, as has been repeatedly stressed, memory is disturbed out of proportion to other cognitive functions. Thus a patient may have been capable of performing adequately the problems posed by a standard intelligence test, and yet, within a few minutes of completing the test, could not recall either the examiner or having taken the test. Although it is recent memory that suffers most, this is a relative matter; the memory of events preceding the illness is always defective and even remote memory may be defective in severe cases.

Of the various abnormalities of memory function, two seem to be of particular importance:

1) A persistent inability to learn newly presented material, i.e. to make "new memories;" and 2) an inability to properly associate past events in their proper temporal sequence.

The inability to learn newly presented material is quite as distinctive of Korsakoff's psychosis as the disorganization of past memory. Since the adaptation to every new situation requires the forming of new memories or at least combining new and old ones, it is the defect in this function which renders the patient helpless in society and capable of performing only the most routine tasks.

Wechsler first emphasized this defect by pointing out that the patients were unable to form new difficult word associations. He regarded this disturbance, rather than the obliteration of old associations, as the central feature of the retention defect in Korsakoff's psychosis. Wechsler attempted to explain the various abnormalities in behaviour and mental function of the Korsakoff patient on this basis. Although this hypothesis may be logically applied to the symptom of confabulation (the patient, unable to recall the appropriate association, responds with the readiest association) it can hardly explain the loss of remote memory.

When the patient attempts to reconstruct the past, there are large gaps in the recounted material. Only isolated facts are retained, and these are not combined in proper chronology, so that the whole is distorted. Or, new material may be introduced, drawn from the patient's own experience and having some logical relation to the story. This defect is particularly characteristic of Korsakoff's psychosis. It becomes obvious after the acute stage of the illness has passed and some improvement in function has occurred, and it remains the dominant disturbance in all but the few patients who make a complete recovery. The disturbance in time sense, the inability to correlate experiences in terms of time relationships, has been considered the fundamental defect in Korsakoff's psychosis.

In addition to the disorders of cognitive function and memory, a significant proportion of patients show a state of confusion, particularly in the early stages of their illness. Such patients are quite unable to grasp their immediate situation — to recognize the examiner as a physician, that they are in the hospital, the time of the day, season of the year, and so forth. They misidentify people around them and misinterpret their actions; they may have periods of apprehension and at times panic in response to their misinterpretations and delusions. In such states, as in delirium, the confusion appears to depend not so much on amnesia as on a widespread perceptual disorder.

No discussion of Korsakoff's psychosis is complete without a statement in respect to confabulation, since this symptom is so widely regarded as a specific feature of this psychosis and as a requisite for the diagnosis. Actually, this symptom is not found in all the patients, and the diagnosis of Korsakoff's psychosis can be readily made without it.

A primary obstacle in the understanding of confabulation is the lack of precise definition and of uniform usage. The non-medical meaning of confabulation is simply to chat or to converse in

a familiar manner. As a symptom of certain forms of insanity confabulation has been defined as "making ready answers and reciting experiences without regard to truth." On the other hand, some authors contend that the confabulatory content always has its basis in past experience; they draw a sharp distinction between this state and what they term fabrication or phantasy, in which the related experiences are purely fictional, emanating from the imagination. Still others draw an analogy between confabulation and fantasy. To some authorities only the spontaneous, self-sustained spinning of a yarn constitutes confabulation, and they reject instances where responses are suggested or provoked by the examiner. Yet such authorities as Pick relate the confabulation to just this latter characteristic—the increased suggestibility of the patient.

Perhaps this matter can be clarified by briefly describing how patients with Korsakoff's psychosis actually perform, using all of the definitions given to the term confabulation rather than arbitrarily acepting any one of them. Only rarely is confabulation a spontaneous phenomenon. Occasionally a casual greeting or a simple question regarding the patient's meals or how he spent the previous day is sufficient to provoke this symptom. Much more frequently, however, confabulation can be brought out by suggestion, such as by a suitably intoned question "Are you sure that you have been here for a week?" Or, "Are you certain you didn't go home yesterday?" It is unusual for a patient to embark on recitation of his experience, each statement apparently nourishing another; usually each response requires another question. The question of whether the recounted material has its basis in truth cannot be answered by a simple affirmative or negative. At one end of the scale are patients, few in number, who spin a yarn without any regard for the truth; more frequently the patient calls up isolated events in his past life, but these are not related in proper temporal sequence. Whether one regards this as memory defect or confabulation is pedantic. If one is not fully aware of the patient's past experiences, the gross temporal dislocations may give them an implausible aspect. But even these statements require qualification: where the content is largely fictional, the general theme of the story may be drawn, in varying degrees, from the patient's experience. Conversely, where related events are apparently drawn from the patient's own past, the embellishment may be fictional. For example, a long-hospitalized patient, on being asked where he spent the preceding day, stated that he went to the cemetery to visit the grave of his dead brother. Actually he had been accustomed to making such visits, the last one about a year before. However, when the patient was forced to explain how he got to the cemetery or how he obtained his clothing in order to leave the hospital, he falsified the answers, to justify his original premise.

What is the factor or factors which determine the development of confabulation? This question does not have a simple answer. It would appear from our observations that in some patients, particularly in the early phases of their illness, confabulation is associated with a state of confusion, the nature of which has already been indicated. Occurring under these circumstances, confabulation can be compared with that which occurs in the course of any delirium, and both probably have their origin in a disorder of perception. The confabulation in this setting tends to improve as the confusion clears. Apart from this group the confabulation is related to the amnestic disorder. Usually such patients show a severe degree of memory disturbance, although in some it may be relatively mild. Also, in general, the confabulation tends to diminish as the memory function improves. The characteristic disorder of retentive memory, in which isolated events are linked without appropriate sequence, logically accounts for confabulation in a large proportion of patients with this symptom. In terms of impairment of other cognitive functions, there is no significant difference between the patients who confabulate and those who do not.

Still unanswered is the question of why one group of patients confabulate and another does not, apparently with similar degrees of confusion, of memory and other cognitive impairment. It has been suggested that the determining factor may be a particular personality pattern. Although it seems logical that the pre-psychotic personality should color the symptoms of a psychosis, no reliable data is available concerning the personality pattern of the patients who develop Korsakoff's psychosis and which, if any, of the personality traits are responsible for the confabulation.

The outcome of this disorder varies. Kraepelin's view, that recovery from Korsakoff's psychosis is a great rarity is quite out of keeping with Korsakoff's original observations, and is no longer held. In a small proportion of patients, complete recovery may be expected. Even when the memory defect and confusion are severe, and confabulation prominent, the prognosis is not hopeless. Complete restoration of memory under these circumstances is unusual; the customary experience is for the patient to make a slow and incomplete recovery over a long period of time. The first sign of recovery may be a difficulty in provoking confabulation—this symptom may disappear within the first weeks of observation, although at times it lingers on for months. Improvement in retentive memory may not become manifest for several weeks or months; once begun, there occurs a slowly progressive restitution of function extending over a year or longer. During this period the patient shows large gaps in memory and the inability to sort out events in their proper temporal sequence; this constitutes a most characteristic feature of the recovery phase in Korsakoff's psy-

chosis. If the patient is seen for the first time during this stage, and if recovery is sufficiently advanced, the connection with "classified" Korsakoff's psychosis may be difficult to discern. In these patients, diagnoses such as "alcoholic deteriorated state" or "organic brain syndrome due to alcohol" are commonly made.

In the light of the preceding descriptions, it is evident that as Korsakoff's psychosis evolves and regresses several abnormal mental states appear. Originally the quiet confusional state of Wernicke's disease and less frequently an associated delirium dominate the scene; later the amnestic-confabulatory state, with a variable degree of confusion becomes prominent; later still, varying degrees of memory defect and the disorder of temporal relationships form the chief mental aberration. Thus it becomes difficult to speak of a classic form of Korsakoff's psychosis; instead, one sees several characteristic forms, which vary with the severity of the disease and the phase of evolution or devolution at the time of the examination.

In concluding the discussion of Korsakoff's psychosis and Wernicke's disease, attention should be drawn to the close relationship between these two syndromes. Cilnically, the majority of patients with Wernicke's disease show signs of Korsakoff's psychosis, either from the time they are first seen, or following a period of apathy and drowsiness. Conversely, the vast majority of patients, in whom an amnestic-confabulatory syndrome is the central feature of the disease, show the stigmata of Wernicke's disease — even years after the onset. Pathologically also, there is a unity of the two diseases. The lesions in the brain are very much the same, whether the patient dies in the acute stages of Wernicke's disease, or in the chronic phase of the illness, when the ocular palsies have cleared and the amnestic symptoms are prominent. It would appear that in the alcoholic, i.e. nutritionally deficient patient, Wernicke's disease and Korsakoff's psychosis represent only different facets of the same disease process.

**Nutritional Polyneuropathy (Neuritic Beriberi)**

The central role played by beriberi in vitamin research and in the development of the modern concept of nutritional disease has been mentioned. This disease was for many years associated only with the Far East. After the first world war, and particularly in the last two decades, it became established there was no essential difference, clinically or pathologically, between the neuropathy of beriberi and that which occurred among alcoholics in the Western Hemisphere. Even the cardiovascular manifestations of beriberi have their counterpart in the alcoholic populations. For these reasons, alcoholic (nutritional) neuropathy and dry (neuritic) beriberi will be treated together.

The symptomatology of nutritional polyneuropathy is remarkably diverse. In fact, many of the patients who came to our attention were asymptomatic, the signs of peripheral nerve affection being revealed only on examination. The majority of these were encountered in the group of patients that has just been described, i.e. the Wernicke-Korsakoff group. The chief abnormalities were thinness of the leg muscles and loss or depression of the knee and ankle jerks, or of the ankle jerks alone. Less frequently there were additional signs such as calf tenderness, somewhat diminished muscle power in the feet and legs or a patchy blunting of pain and touch sensation over the feet and shins.

The majority of patients with signs of polyneuropathy also suffer of neuritic symptoms —weakness, parasthesias and sometimes pain. The symptoms are usually insidious and slowly progressive, although at times there may be a sudden increase in weakness. In a small but characteristic group the transition from an asymptomatic state to one of virtual paralysis occurs in a matter of several days. The symptoms are at first referred to the distal portions of the limbs and progress proximally if the illness remains untreated. The legs are affected earlier than the arms, sometimes exclusively, although in exceptional cases the arms may be affected more severely than the legs. Motor and sensory symptoms occur concomitantly, although the patient may complain much more of one than the other; usually the motor disability constitutes the chief complaint. Sensory symptoms consist mainly of numbness, prickly feelings, coldness, deadness, tenderness of the calf and plantar musculature, or unusual sensitivity to contact. In a minority of patients, pain and parasthesias constitute the chief complaints. The pain may take the form of a dull constant ache in the feet, sometimes of the entire leg; often the pains are sharp and lancinating, momentary in duration, quite like the lightning pains of tabes dorsalis. Complaints of coldness are common, but these are purely subjective, the feet feeling quite warm to touch. Much more distressing and incapacitating are the "burning" feelings and sensations of heat; usually these affect the soles, less frequently the dorsal aspects of the feet as well; they fluctuate in intensity or may be clearly intermittent in nature. Characteristically, a patient afflicted with pains and parasthesias suffers not one, but all of the symptoms enumerated. Although the painful symptoms may arise spontaneously, they are made much worse by a contactual stimulus. The amount of pressure required to produce discomfort varies; in severe cases the patient cannot bear to have the bedclothes touch his feet or to pick up a utensil; or he may be unable to walk despite the preservation of motor power.

Examination reveals varying degrees of motor, reflex and sensory loss. As the symptoms would suggest, the signs are symmetrical, usually more prominent in the distal portions of the limbs, and often confined to the legs. In rare instances they are more prominent in the arms than in the legs,

and more prominent in the proximal than in the distal musculature of the leg. The weakness varies greatly in degree — it may be evident only with muscular exertion or it may take the form of a foot or wrist drop or even a complete paralysis of the limb. Deep reflexes in the legs are almost universally lost, even with the mildest degrees of weakness. In the arms this usually applies as well, although occasionally the tendon reflexes are retained despite serious loss of power in the hands. In a small number of patients, particularly those with pain and parasthesias, the reflexes may be of greater than average briskness.

Sensory loss usually involves all the modalities. Although one cannot adequately equate touch, pain and temperature, vibratory and position sense, some patients seemingly show an impairment or loss of one modality out of proportion to the others. No consistent pattern has evolved, however, which would enable one to predict, from the patient's symptoms, which mode of sensation might be disproportionately affected. There is no sharp border between normal and impaired sensation; the sensory loss, which is most profound distally, shades off gradually, the transition to normal sensation occurring over a long vertical extent of the limb.

As a rule, only the limbs are affected and the abdominal, thoracic and bulbar musculature are intact. In some instances of oriental beriberi, sensory loss has reportedly involved the face and abdomen as well. Tinnitus, vertigo, nerve deafness, aphonia due to vocal cord paralysis (particularly in infants), and retrobulbar neuropathy may also complicate beriberi in rare instances. The relation of these disturbances to beriberi has been a point of contention that cannot be settled with finality, since the specific cause of either is not known. Far more frequently they complicate the syndrome of ataxia, and burning, tender feet and are therefore appropriately considered as a part of Strachan's syndrome.

The spinal fluid in the nutritional neuropathies is usually normal, although an occasional case may show a slight elevation of the protein content. The normal spinal fluid may be helpful in distinguishing the rapidly evolving form of nutritional neuropathy from infectious polyneuritis.

Recovery is invariably a slow process. In the mildest cases there may be considerable restoration of motor power in a few weeks; in the severest forms several weeks may pass before even the first signs of recovery become manifest, and up to a year before the patient is able to walk unaided. Recovery in severely affected patients is often incomplete, and they may be left with some weakness of the feet and a lasting abolition of the knee and ankle jerks. The abandonment of therapy either greatly prolongs recovery or seriously disables the patient by contractures of the limbs.

Pathologically, nutritional neuropathy is characterized by a degeneration of the peripheral nerves, and in advanced cases of the anterior and posterior nerve roots. The degenerative process is more intense in the distal segments than in the proximal ones. Both the myelin and the axis cylinders are destroyed, the former probably earlier and to a greater extent than the latter. The degeneration is Wallerian in type or there may be segments of nerve in which the myelin is lost and the bare axis cylinders remain (the segmental demyelination of Gombault). Dorsal root ganglion cells may be lost to variable extent, and the anterior horn cells of the spinal cord show an "axonal reaction." The latter change is probably secondary to the axis cylinder damage in the anterior roots and peripheral nerves.

**The "burning feet" syndrome.** The term "burning feet" is frequently applied to a state in which pain in the extremities is the outstanding symptom and in which the advanced signs of neuropathy may be absent. It is doubtful whether this syndrome merits classification apart from other nutritional neuropathies; it is here considered separately out of deference to the many reports from the prisoner-of-war camps of the Far East. In these reports the pain is variously described as tingling, burning, aching, shooting, cramp-like, or resembling the lightning pains of tabes. The pain was often very severe, greatest at night and interfered with sleep. Some patients found relief from the application of cold; others only in movement. The presence of associated neuritic signs was a variable matter. In some patients, wasting, dropped foot, reflex loss and sensory changes were completely wanting; in a significant proportion the tendon reflexes were exaggerated, but without clonus or extensor plantar responses. However, in other groups of patients, the painful feet were but one stage in the evolution of a peripheral neuropathy characterized by tenderness of the calves, reflex and sensory loss and ataxia, and complicated in many cases by retrobulbar neuropathy.

In alcoholic (nutritional) polyenuropathy, pain is the outstanding symptom in a small group of patients; however, they do not constitute a distinct group in terms of their neurological signs. Pain and dysesthesia may be a prominent symptom in cases with all degrees of motor, reflex and sensory loss. In some cases the weakness may be slight or absent, and in rare instances reflexes may be retained despite a severe "burning feet" syndrome. However, in all cases of severe "hyperesthesia," even where the slightest stimulus appears intolerable, there is usually sensory loss; one is able, by using finely-graded stimuli, to demonstrate an elevated threshold to painful, thermal, and tactile stimuli in the "hyperesthetic" zone. The term "hyperesthetic" is, therefore, not well chosen; it implies a heightened receptiveness of the nervous system or an increased response of the receptors to tactile and painful stimuli. Actually, there is an underlying sensory deficit or an elevated threshold

to various stimuli; once the stimulus is perceived, however, it may have a severely painful or unpleasant quality (hyperpathia).

The term "burning" is not particularly applicable, considering the wide variety of symptoms apart from thermal dysesthesias. Because of this, as well as the fact that the hands may be involved, the term "painful extremities" is preferred by some.

Although undoubtedly nutritional, the specific deficiency responsible for the dysesthesias has not been clearly established. The pathophysiology is likewise unknown. Spillane suggests that this affection is an early stage of nutritional disturbance in the nerves to the lower limbs. He draws an analogy to the burning pains produced by the interruption of conduction in large nerve fibres by rendering a limb ischemic. We have been impressed by the similarity of the pain to causalgia and in several patients have succeeded in abolishing the pain for several hours to days by paravertebral sympathetic block. These observations require confirmation.

**Nutritional Amblyopia**

This term refers to the visual failure which occurs in nutritional disease but which is not due to a lesion of the cornea or other parts of the eye concerned with refraction. The site of the lesion is most likely in the optic nerve, so that the term "retrobulbar neuropathy" is a suitable synonym.

Clinically the characteristic symptom is a blurring of vision for near and distant objects, usually developing gradually over a period of several weeks. Examination discloses a reduction in visual acuity, the presence of central and centrocecal scotomata for white or colors, and, in some cases, fundoscopic abnormalities. The latter vary from a mild papillitis with slight hyperemia and perhaps blurring of the disc margins to pallor of the optic disc in the most advanced cases. Retinal hemorrhages may be seen occasionally. These changes are always bilateral, though frequently asymmetrical. Untreated, the disease may progress to irreversible optic atrophy. With nutritious diet and multiple vitamin supplements, improvement occurs in all instances, though to a variable extent.

In this country, many, if not all, of the cases of retrobulbar neuropathy attributed to the toxic effects of alcohol or tobacco are probably of nutritional origin. The clinical experiments of Carroll have convincingly demonstrated that recovery from amblyopia follows improvement in nutrition, despite continued indulgence in alcohol and tobacco. Retrobulbar neuropathy may occur as the only manifestation of deficiency, but far more frequently it is combined with other nutritional syndromes. Most of our patients with amblyopia also showed signs of peripheral neuropathy; in addition, a small number of cases were complicated by the Wernicke-Korsakoff syndrome and a similarly small number by a spinal spastic and ataxic syndrome.

Deficiency amblyopia was particularly prevalent during the last war in the prisoner of war and civilian internment camps of the Far East. Although it had previously been described in association with beriberi and pellagra, the main wave of incidence did not coincide with either of these syndromes, but with the orogenital and "burning feet" syndromes. Although the amblyopia seemed to respond best to a diet rich in riboflavin, the etiological relationship to this vitamin cannot be regarded as established.

There are no complete pathological studies of deficiency amblyopia. Fisher has examined the optic nerves of four Canadian prisoners of war with residual impairment of visual acuity. The nerves showed a loss of myelin and nerve fibres in the region of the papillomacular bundle, the fibres from the periphery of the retina appearing quite healthy.

**Pellagra**

Pellagra, in its fully developed form, is characterized by dermatitis, diarrhea, mouth lesions, mental and nervous symptoms. When this central core of symptoms is present, the clinical state is readily recognized. Judging from the literature, however, the symptomatology is so heavily endowed with all manner of para-phenomena, that the definition of its boundaries is quite artificial at the present time.

Here we shall be concerned only with the neurologic manifestations, which in themselves are extremely diverse. Pellagra is essentially an encephalopathy, although involvement of all parts of the nervous system has been described. In the early stages of the disease the symptoms are mainly mental, and may be mistaken for those of a psychoneurosis. Insomnia, fatigue, nervousness, irritability or feelings of depression are common complaints. Examination may reveal retardation of mental processes and impairment of memory. Sometimes an acute confusional psychosis dominates the clinical picture. Pellagra may not only produce insanity, but occasionally may result from it, by virtue of the anorexia and refusal of food that accompanies certain mental illnesses. The manifestations of spinal cord involvement have not been clearly delineated, perhaps because the patient's mental state precluded accurate testing. In general, they are referable to both the posterior and lateral columns, predominantly the former. Neuritic signs are the least common of the neurological manifestations, and from the descriptions are indistinguishable from those of neuritic beriberi. Signs such as tremors, extrapyramidal rigidity, sucking and grasping reflexes, and coma have been ascribed to the pellagrous syndrome, as have various disorders of the special senses.

The distinctive pathological changes in pellagra are most readily discerned in the large cells of the motor cortex, and cells of Betz, although the same changes are seen to a lesser extent in the smaller pyramidal cells of the cortex, the large

cells of the basal ganglia, the cells of the cranial motor nuclei and the dentate nucleus, and the anterior horn cells of the cord. The affected cells appear swollen and rounded, with eccentric nuclei and loss of the Nissl particles. This change was first described by Adolf Meyer as "central neuritis" and is frequently referred to as "axonal reaction" because of the similar nerve cell change which occurs most frequently in the anterior horn cells when their axones are severed. It has never been decided whether or not the central neuritis of pellagra is the same as the axonal reaction and whether it is, in fact, dependent on injury to the axones of the Betz cells. One argument against this possibility is the frequency with which the motor tracts are interrupted in the brain and spinal cord without an axonal reaction appearing in their cells of origin. Moreover, in pellagra the cortical nerve cell changes have not always been associated with disease of the pyramidal tracts. It is more likely that this represents a primary cytologic degeneration of the whole motor cell; or it may represent a cytoplasmic alteration to a purely biochemical abnormality in the axone which only reaches the stage of visible myelin degeneration in certain of the more acute or severe cases.

There is considerable disagreement in the literature concerning the spinal cord changes in pellagra. Some authors are of the opinion that they do not differ radically from those of subacute combined degeneration, i.e. a patchy, non-systematized degeneration in the posterior and lateral funiculi. Most commonly, however, the spinal cord lesions are described as a symmetrical degeneration of the dorsal columns, especially of Goll, and to a lesser extent of the pyramidal tracts. The posterior column degeneration affects specific system of fibres and is secondary to the degeneration of the posterior roots. The nature of the pyramidal tract lesion in pellagra is not known; one can only speculate that this change is secondary to the pyramidal cell degeneration.

A spinal spastic syndrome, apart from the other symptoms and signs of pellagra, may be a prominent manifestation of deficiency disease. The chief clinical signs are spastic weakness of the legs with absent abdominal and increased tendon reflexes, clonus, and extensor plantar responses. These signs rarely occur alone, however. In nutritionally depleted alcoholics, in whom pyramidal tract signs are very rare, they have occurred together with Wernicke's disease, delirium, and retrobulbar and peripheral neuropathy. In prisoner of war camps the "spastic syndrome" was accompanied by mental and emotional changes, dimness of vision, and at times widespread muscular rigidity, delirium, coma and death. The pathological changes in this syndrome are not known, so that accurate classification is difficult. Although it has been described under the term "pellagrous encephalopathy," many of the classical signs of pellagra are lacking, and epidemiologically, this syndrome occurred after the large outbreaks of Wernicke's disease and pellagra.

**Strachan's Syndrome**

Not all of the nutritional disorders of the nervous system fall into the readily recognized clinical categories of the Wernicke-Korsakoff syndrome, beriberi or pellagra. Beginning with the report of Strachan in 1888 and culminating with the recent observations among prisoners of war and civilian internes, there have appeared a large number of reports which cannot be forced into the boundaries of the classical nutritional syndromes. To derive a unified concept from this literature is a bewildering task; the clinical descriptions are far from adequate and pathologic studies are virtually non-existent. The fact that more than one portion of the nervous system is involved and that each of these parts may be involved in varying degrees of severity has created an infinite variety of seemingly unconnected diseases. Nevertheless, when these descriptions are viewed collectively, a symptom-complex emerges, the principal features of which are parasthesias of the feet, hands, trunk and even face, burning, tender feet, impaired vision and loss of reflexes. Less common features are dizziness, deafness, hoarseness, acute myasthenia, spasticity and mucocutaneous lesions. To this syndrome the term Strachan's syndrome is here arbitrarily applied; this is done for the sake of brevity and because Strachan was perhaps the first to describe this syndrome, even though he did not recognize its nutritional etiology.

Strachan's syndrome is essentially a disorder of the peripheral and optic nerves. Clinically, sensory symptoms and signs dominate the picture, in this respect differing from beriberi. Parasthesias of the extremities, face and trunk, painful "hyperesthesia" of the feet, loss of superficial and deep sensation and ataxia are the common manifestations. On the other hand, foot drop and muscle weakness occur very rarely. A frequent associated disorder is failing vision, which may go on to complete blindness and pallor of the optic discs. In general, deafness and vertigo are rare complications, but in some outbreaks these symptoms were so common as to earn the epithet "camp-dizziness." Along with the neurologic signs there are varying degrees of stomato-glossitis, corneal degeneration and genital dermatitis; these mucocutaneous lesions are often spoken of together as the "oro-genital" syndrome, and are quite distinct from those of pellagra.

There have been only a few pathologic studies of this syndrome. Aside from the damage to the papillomacular bundle in the optic nerve, which has already been described, the most consistent abnormality has been a demyelination in each column of Goll adjacent to the midline. This indicates a systemitized degeneration of the central process of the bipolar sensory neurons of the

lumbosacral spinal ganglia. The fact that the primary sensory neurone is the chief site of disease is consistent with the predominant sensory symptomatology.

### Subacute Combined Degeneration of The Spinal Cord

Subacute combined degeneration of the spinal cord, the neurologic component of pernicious anemia, is an example of a single vitamin deficiency (vitamin $B_{12}$), but its nature is clearly different from the nutritional diseases that have just been considered. Castle has aptly summed up its unique features as follows: "Pernicious anemia is usually an example of a highly specific isolation of the affected person from his alimentary environment. Thus, this disease would not develop if the patient could effect daily the transfer of a millionth of a gram of vitamin $B_{12}$ the distance of a small fraction of a millimeter across the intestinal mucosa and into the blood stream. This he cannot do, principally as a result of failure of his stomach to secrete into its lumen some essential but still unknown substance. Yet the patient may each day absorb without much difficulty the products of the digestion of many grams of carbohydrate, fat or protein from foods that in addition may contain consequential amounts of vitamin $B_{12}$ in terms of his trivial need. Such starvation in the midst of plenty has elsewhere been called "conditioned deficiency disease."

Lichtheim in 1887, followed by his pupil Minnich, were the first to describe the spinal cord disease accompanying pernicious anemia. In the decade that followed a large number of papers appeared which clearly delineated the main clinical and pathologic attributes of the neurologic disease as we know them today. The intimate relationship of the spinal cord disease to pernicious anemia, however, was debated for many years. The relationship was denied by some of the most authoritative English authors, and only following the writings of Bramwell and of Woltmann, who stressed the high statistical coincidence, and of Hurst and Bell who emphasized that gastric achlorhydria was invariably present in both, was their unity fully appreciated.

**Clinical Manifestations of Subacute Combined Degeneration of Cord.** Symptoms of nervous system disease are present in a high proportion of patients with pernicious anemia, varying from 75 to 89 per cent according to different authors. In general, the neurologic symptoms and signs have a uniform mode of onset and progression. In practically all instances the patients first notice general weakness and paresthesias. Of tne latter, tingling or "pins and needles" feelings are the most common, to be followed in frequency by a large variety of other peculiar sensations, often vaguely described, such as numbness, stiffness, deadness, tightness, feelings of heat or cold, formication and shooting pains. The paresthesias tend to be constant, to progress steadily, and to be the source of much distress. They are usually localized to the distal parts of all four limbs in a symmetric distribution. The lower extremities are as a rule involved before the upper ones. Less often vague aches and pains, girdle sensations, cramping of the calves and even more rarely, bladder and bowel disturbances are the initial complaints; or there may be a disorder of the special senses, such as diminution of the sense of smell, or very rarely of visual acuity, perversions of taste, or roaring or thumping noises in the ears. As the illness progresses stiffness and weakness of the limbs develops, especially of the legs, and this, combined with a defect in postural sensation, produces a weak unsteady gait and awkwardness of the limbs. These symptoms, if untreated, may progress, and in their more advanced state take the form of an ataxic paraplegia with variable degrees of spasticity and contracture.

Early in the course of the illness, when only paresthesias are present, there may be no objective signs. Later, the neurologic examinations may indicate a disturbance of all parts of the nervous system, but mainly of the posterior and lateral columns of the spinal cord. Of the signs of posterior column affection, loss of vibration sense is by far the most consistent; this is more pronounced in the legs than in the arms and frequently the loss of vibration sense extends over the trunk, leaving little doubt that the nervous disorder is located in the posterior columns of the cord and not in the spinal roots or peripheral nerves. Position sense is involved somewhat less frequently, and in rare instances may even be more or less intact despite impairment of vibration sense. Characteristically, there is greater impairment of deep than of superficial or cutaneous sensation, but the latter is by no means always spared. Isolated instances of loss of pain, temperature and tactile sensation below a segmental level on the trunk do occur, implicating the spinothalamic tracts, but such a finding should always suggest the possibility of some other disease of the spinal cord. More commonly the defect of superficial sensation takes the form of a mild blunting of touch, pain and temperature sensation over the limbs in a distal distribution.

Examination of the motor system discloses loss of power, spasticity, changes in the tendon reflexes, clonus and extensor plantar responses. Except in the most advanced cases these signs are practically limited to the legs, their prominence depending on the degree of corticospinal involvement. At first the patellar reflexes are found to be diminished in activity as frequently as they are increased, and may even be absent; the Achilles reflexes are more frequently depressed than hyperactive. With treatment the absent or depressed reflexes may return to normal or even become hyperactive. The abnormality of gait is governed by the relative amount of damage to the posterior and lateral columns of the cord and may therefore be pre-

dominantly ataxic or spastic, usually both. The nervous system involvement in subacute combined degeneration is characteristically although not always symmetric. A definite asymmetry of motor or sensory findings maintained over a period of weeks or months should always cast doubt on the diagnosis of subacute combined degeneration of the spinal cord.

Mental signs are frequent, ranging from irritability, apathy, somnolence, suspiciousness and emotional instability, to a marked confusional or depressive psychosis, or intellectual deterioration. Signs of visual impairment are distinctly rare and, when present, take the form of centrocecal scotomas. If involvement of the optic nerve is severe, optic atrophy may occur.

**Neuropathologic Changes in Subacute Combined Degeneration of Spinal Cord and Brain.** The clinical signs are readily explained by the pathologic lesions of the nervous system, which have a characteristic appearance and a regular distribution. The pathologic process takes the form of diffuse, although uneven, degeneration of the white matter. There are multiple foci of spongy degeneration, often in relationship to small blood vessels and varying from 0.1 to 2.0 mm. in diameter. The myelin sheaths and the axis cylinders are both affected, the former perhaps earlier and to a greater extent than the latter. There is relatively little fibrous gliosis, except in treated cases in which destructive cord lesions existed prior to adequate therapy. In early cases only the posterior columns are affected and in more advanced cases the lesions here appear older. The changes most often begin in the posterior columns of the thoracic cord. It would appear that they spread from this region up and down the cord as well as forward into the lateral columns. The lesions are not limited to specific systems of fibers within the posterior and lateral funiculi but are scattered irregularly through the latter. The paresthesias, impairment of vibratory and position sense, ataxia and the Romberg sign are due to affection of posterior columns, and lesions here may also account for loss of tendon reflexes. Weakness, spasticity, increased tendon reflexes and Babinski signs depend on involvement of the pyramidal tracts in the lateral columns. The spinothalamic tract may be involved in the pathologic process, which explains the occasional clinical finding of loss of pain and temperature sensation at a segmental level on the trunk. In advanced forms of the disease similar pathologic changes may occur in the white matter of the brain. Patients who disclose such lesions at postmortem examination will have shown mental symptoms during life, although cerebral lesions are found only infrequently in the cases in which mental symptoms were prominent. Instances of degenerative lesions of the optic nerves have also been verified at necropsy.

There is no unanamity of opinion regarding peripheral nerve lesions in pernicious anemia. The clinical evidence of peripheral nerve involvement is stronger than the pathologic and the existence therefore of disease in the spinal roots and nerves is largely inferential. It has been suggested by some authorities that reversibility of neurologic signs is indicative of peripheral nerve involvement. The distal and symmetric blunting to pain, touch and temperature that occurs in many cases is certainly a point in favor of peripheral nerve disease. The pathologic reports are not altogether convincing. Greenfield and Carmichael, by making counts of nerve fibres in cross sections of a digital nerve of the foot, have shown that there is a loss of myelin without corresponding loss of axis cylinders.

Aside from the characteristic laboratory findings of pernicious anemia, there are few laboratory tests that are specifically helpful in the diagnosis of the nervous system disorder. The spinal fluid is usually normal, although occasionally there may be a slight elevation of the protein content. There are reports of an electroencephalographic abnormaity consisting of a diffuse slow-wave activity in many cases of pernicious anemia. This bears no relation to the age of the patient, the severity of the anemia or the presence of neurologic symptoms. The authors of these reports conclude that the changes in the electroencephalogram are due to a specific defect in cerebral metabolism.

**Efficacy of Liver and Vitamin $B_{12}$ in Subacute Combined Degeneration.** Therapeutic experiments have yielded much information concerning the cause and mechanism of both hematologic and neurologic components of the disease. At first it was believed that the spinal cord and blood changes were due to separate deficiencies and for this reason treatment with liver extract was supplemented with crude liver. However, there is now sufficient evidence that the response to vitamin $B_{12}$ alone is in all ways comparable to that from refined liver extract or crude liver, and that all the symptoms are due to deficiency of vitamin $B_{12}$. It has been sugested that the equivalent of 1 microgram of parenterally administered vitamin $B_{12}$ daily may be an adequate dosage in the treatment of subacute combined degeneration of the cord, but in practice the amount prescribed is usually higher.

With the introduction of liver therapy the prognosis in subacute combined degeneration of the cord was radically altered. This was not immediately apparent, and in the early days of liver therapy it was thought that spinal cord symptoms could actually develop during liver therapy or progress despite treatment. Others recognized that weakness and mental signs improved with liver therapy but they supposed that this was the result of improvement in general health and of the anemia.

With the further study of cases without anemia and the development and use of refined liver

fractions, the salutary effects of treatment on the nervous symptoms became obvious. The improvement in neurologic signs has been shown to be quite dramatic and complete when effective therapy is instituted early after the onset of symptoms. In practically all instances at least partial improvement is effected, although in long-standing cases often the best that can be accomplished is arrest of progression. According to Ungley, the most important factor influencing the response to treatment is the duration of the disease as measured by the duration of difficulty in walking. The greatest improvement occurs in those whose difficulty in walking is of less than three months' duration; and, conversely, the least improvement occurs in those with difficulty in walking of longer than two years' duration. Factors of little or no importance are age, sex, severity, arteriosclerosis, hypertension and the degree of anemia. Neurologic relapses during therapy are usually associated with infections and can be corrected by increasing the dose of liver extract or vitamin $B_{12}$. Ungley finds that all neurologic symptoms and signs may be improved; that extensor plantar responses are as reponsive to treatment as paresthesias and loss of vibratory sense. The return of absent deep reflexes is commonly observed, although at times it may take longer than a year. Most improvement occurs during the first three to six months of therapy, but there may be slight improvement after that for several months, or a year or longer.

**Pathogenesis of Pernicious Anemia and Subacute Combined Degeneration.** The modern concept of pernicious anemia dates from 1926 when Minot and Murphy demonstrated the efficacy of liver in the dietary treatment of the disease and thus offered the first convincing evidence that pernicious anemia, with its accompanying neurologic lesions, was due to a nutritional deficiency. In the years that followed, oral liver therapy was succeeded by parenteral treatment with purified liver extracts and in 1948 the component of liver specifically active in pernicious anemia, vitamin $B_{12}$, was isolated. This red, cobalt-containing vitamin was shown effectively to correct the hematologic and neurologic disorders of pernicious anemia when administered in only microgram doses.

Shortly after the discovery of liver therapy Castle offered a hypothesis of the pathogenetic mechanism. He suggested that an essential material (extrinsic factor) from the diet interacted with a constituent of normal gastric juice (intrinsic factor) and gave rise to an active erythrocyte-maturing factor. He also conclusively demonstrated that it is precisely this lack of intrinsic factor in the meager gastric secretion that characterizes the patient with pernicious anemia. The exact nature of the intrinsic factor has not yet been defined, although potent purified intrinsic factor concentrates derived from hog duodenal mucosa have been prepared. On the other hand, the extrinsic factor is now known to be vitamin $B_{12}$ and it appears to be identical with the erythrocyte-maturing factor derived from liver. With present knowledge then, Castle's theory has been modified only slightly and pernicious anemia is now regarded as a conditioned nutritional deficiency in which, owing to a lack of intrinsic factor, dietary vitamin $B_{12}$ is not absorbed and is therefore not available for normal metabolism. The precise mechanism whereby intrinsic factor effects the transfer of vitamin $B_{12}$ to the tissue remains obscure but most likely its function is to facilitate the absorption of vitamin $B_{12}$ from the upper gastrointestinal tract. Once within the body, vitamin $B_{12}$ is freely transported in the serum and is available for hematopoiesis and for maintenance of the integrity of the nervous system.

Other specific nutrients have been shown to be temporarily effective in correcting the hematologic defect in pernicious anemia. Of these folic acid and its metabolically active form, the citrovorum factor, have been most extensively studied. Although it was immediately evident that the anemia responded to the administration of folic acid, subsequent studies clearly indicated that it was not the extrinsic factor. But, what is most important, folic acid does not prevent the development nor does it arrest the progression of the neurologic disease in pernicious anemia. In fact, it may accelerate the development of nervous system lesions.

By inference from microbiologic investigation, and to some extent from clinical studies, it would seem that the role of vitamin $B_{12}$ and folic acid in hematopoiesis is concerned with nucleoprotein synthesis. This explains the interchangeability of these two vitamins in hematopoiesis. It has been suggested that folic acid fails to maintain the integrity of the nervous system because it functions in desoxyribonucleic acid synthesis and is not concerned with ribonucleic acid formation, as is vitamin $B_{12}$. The apparently deleterious action of folic acid upon subacute combined degeneration is still a matter for speculation but may be due to a mass action effect accelerating development of absolute vitamin $B_{12}$ depletion, the result of altered kinetics of vitamin $B_{12}$ utilization or storage, or simply to the creation of vitamin imbalance by treating only part of the existing deficiency state.

**Clinical Problems.** Subacute combined degeneration of the spinal cord still poses many clinical problems, chiefly concerned with making an early diagnosis. The main reason for this difficulty is the lack of parallelism between the hematologic and neurologic manifestations. With the widespread use of folic acid this problem has become more acute, since this drug may cause a hematologic remission for an indefinite period, while the neurologic signs worsen, often to an irreversible stage. Other problems concern the difficulty of distinguishing between intrinsic spinal

cord disease of non-pernicious anemia type from posterior and lateral column disease due to pernicious anemia; and of distinguishing pernicious anemia and subacute combined degeneration from other macrocytic anemias and their associated neurologic disturbances.

In most instances the diagnosis of subacute combined degeneration of the cord can be made by utilizing the standard methods for the diagnosis of pernicious anemia — the examination of the blood, gastric acidity and the bone marrow. These methods are of limited value when the anemia is mild or absent or when the anemia has been corrected by folic acid. A therapeutic trial of vitamin $B_{12}$ may be employed, but recourse to such a procedure means that therapy must be continued indefinitely, and the diagnosis may remain in doubt. Under these circumstances a number of refined diagnostic aids may be employed, such as the microbiologic assay of $B_{12}$ utilizing the organism Euglena gracilis, and the measurement of intrinsic factor activity by the hepatic deposition or urinary excretion of radioactive vitamin $B_{12}$.

# Neurosurgery

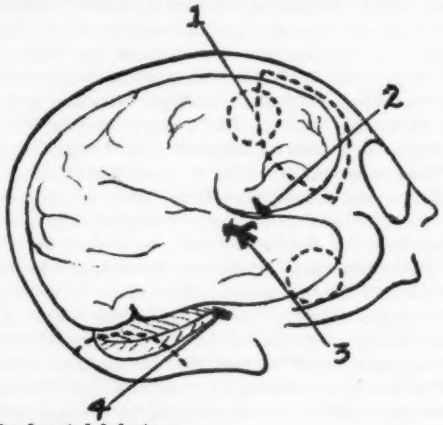
## Neurosurgical Procedures for the Relief of Intractable Pain

Harold N. Lynge, M.D. and Gerd Fischer, M.D.

There is a tendency among some to look upon the specialty of neurosurgery as a field dealing only with hopeless lesions, necessarily involving a very high rate of mortality and when not ending in death at best leaving the luckless patient in a crippled state mentally or physically or both. This is not at all the true state of affairs and in, for instance, the neurosurgical management of intractable pain, as in many other problems, a great deal can be done to relieve the patient's suffering and still leave him relatively intact.

The first group of procedures to be described are intracranial in nature and we shall first mention prefrontal lobotomy. It is not our intention to dwell on the technical aspects of this or other less well-known procedures related to it, such as topectomy, postcentral gyrectomy, etc. Suffice it to say that prefrontal lobotomy is designed to interrupt the frontothalamic fibres by severing them with a leukotome through trephine openings placed lateral to the longitudinal sinus and just anterior to the coronal suture (see diagram No. 1). The procedure can be useful in some cases where pain caused by inoperable carcinoma or proved metastases is no longer bearable; lobotomy does not result in lack of sensation but the psychic reaction to the painful stimulus is absent and there is a lessening of anxiety and distress which is usually present pre-operatively because of the patient's knowledge that he has incurable cancer.

A comparatively new procedure in the neurosurgical field is the operation of hypophysectomy. This was first done by Olivecrona of Stockholm in 1952 in the therapy of carcinoma of the breast with skeletal metastases and resulting pain. Several reports have emphasized the relief of pain following this procedure in addition to the retardation of growth of the malignant lesion. The operation is not particularly formidable; a right transfrontal flap (see diagram No. 2) is turned as for pituitary tumors, but it is a simpler operation than the removal of a pituitary adenoma where adjacent structures and pressure relationships within the cranial cavity are disturbed.




1. Prefrontal lobotomy
2. Hypophysectomy
3. Sectioning of sensory root of Vth nerve
4. Sectioning of glossopharyngeal nerve

Another intracranial procedure for the relief of pain is much more widely known—the section of the posterior root of the Vth cranial nerve for trigeminal neuralgia. This is done (as shown in the diagram No. 3) via the temporal route; again we merely mention the newer modifications of operative procedure in the control of this malady, namely the "trigeminal decompression" (which has already shown a high percentage of recurrences) and the "trigeminal compression" operation. Some of the most grateful patients are those who have been relieved of their trigeminal pain by section of the sensory root of the Gasserian ganglion.

Another extremely painful malady, and one very similar to trigeminal neuralgia but fortunately much less common, is glossopharyngeal neuralgia; this follows the distribution of the 9th cranial nerve and the patient can be permanently relieved of the pain by intracranial section of the nerve, using a unilateral suboccipital approach (see diagram

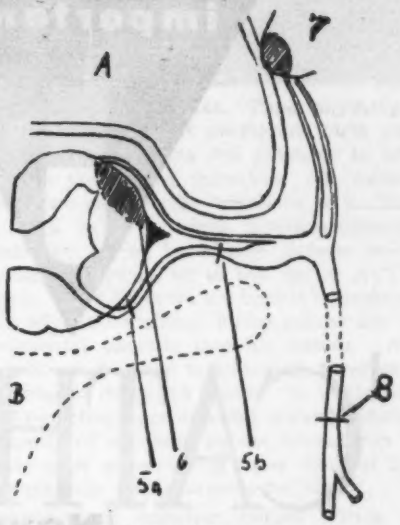
No. 4). It should be mentioned here that intracranial section of the Vth and IXth nerves is sometimes done together (also through the same suboccipital approach) for relief of intractable pain in the face and neck resulting from carcinoma of the jaw. In combination with the above procedure and depending upon how much involvement there is, it is sometimes necessary to section the posterior roots of the upper cervical nerves intraspinally, which brings us to the second group of procedures, namely the intraspinal operations for the relief of pain.

In such conditions as occipital neuralgia and intercostal neuralgia, rhizotomy frequently has to be resorted to. Although occipital neuralgia involves chiefly the 2nd cervical nerve on either side, there are times when the 1st and 3rd cervical nerves are also involved and in this case, intraspinal rhizotomy of the sensory components of all three nerves is required for complete relief. Intercostal neuralgia may accompany or follow herpes zoster or may develop independently of skin eruptions. It usually involves two or three dermatomes, and a unilateral laminectomy with rhizotomy of the sensory roots frequently has to be resorted to.

Rhizotomy of the sensory roots of the brachial plexus has been employed for metastatic lesions of the axilla and brachial plexus. While on the subject of rhizotomy we might mention that it can be intradural or extradural (see diagram No. 5a and No. 5b), depending upon certain conditions; in the sacral region it is best to use the intradural method in order to assure preservation of the anterior root.

A very valuable procedure for the relief of pain is the spinothalamic tractotomy or chordotomy. The thoracic chordotomy is most prevalent (see diagram No. 6); it is usually performed between the 1st and 3rd thoracic segments to relieve pain on the opposite side of the body at a level below the 7th or 8th thoracic segment. It is often done bilaterally for intractable pain due to inoperable and metastatic lesions of the legs, pelvis, spinal cord or spinal column below the diaphragm. High cervical chordotomy is being done more and more; when this procedure is performed at the level of the 1st or 2nd segment it is useful in relieving pain in the upper extremities, and the level of hypalgesia is often more consistent than when done at the thoracic level. However, bilateral high cervical chordotomy is not advisable.

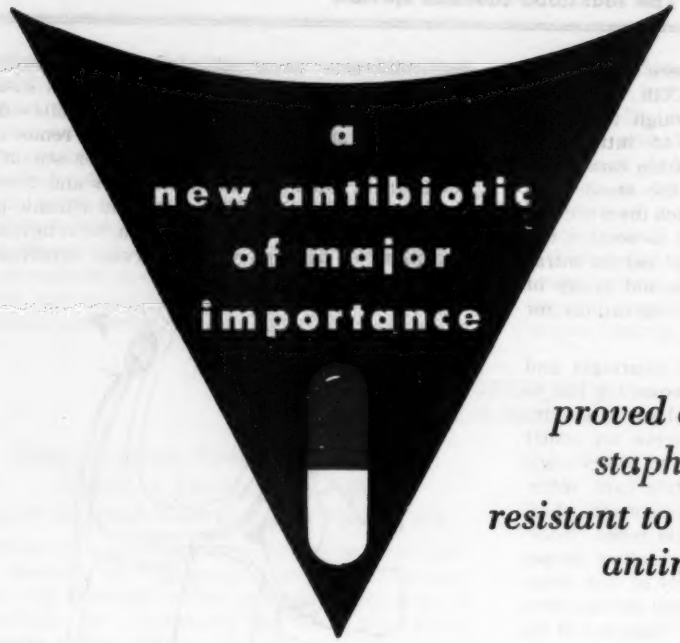
We now come to operations designed for relief of such conditions as causalgia, Sudek's atrophy, visceral pain, and so on. In these conditions, the pain pathway is in the sympathetic nervous system, and consequently interruption of the appropriate pathway (see diagram No. 7) can relieve the pain. The burning feeling and hypersensitivity characterizing causalgia of the hand can be relieved by cervicothoracic preganglionic or postganglionic sympathectomy. The pain of Raynaud's disease and Thromboangiitis obliterans can be relieved by resection of the sympathetic trunk and removal of ganglia; this interrupts the vasomotor stimuli to the arteries supplying the extremities and thereby decreases the spasm. The pain from chronic pancreatitis and pancreatic calculi can be relieved by resection of the splanchnic nerves, involving a thoracolumbar approach.




5a Intradural rhizotomy
5b "extradural" rhizotomy
6. chordotomy
7. sympathectomy
8. Neurectomy
A. Vertebral body
B. Spinous process and lamina

The last procedure for the relief of pain which we shall mention is neurectomy (No. 8 in diagram). This should be done only in selected cases. Intractable pain in the hip joint can be alleviated by section of the obturator nerve, and section of the sciatic sensory rami has also been done to denervate the hip. The reason that such procedures should be undertaken only in certain cases is that pain is not the only sensory modality lost but also touch and proprioception.

We have touched upon the commonest and, what we consider to be, the most efficacious neurosurgical methods for the alleviation of intractable pain. There are a few others which we have not mentioned because they are too hazardous in some instances and in others have proved unworthy. The procedures mentioned are not dangerous and if the patient has a prognosis of more than a few months of life we feel he should be given the advantage of freedom from pain and yet be spared the ignominy of becoming a narcotic addict.

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# Children's Hospital, Winnipeg

## Infections in the Newborn

**Howell Wright, M.D.**
**Professor of Pediatrics, University of Chicago Medical School**

The process of birth removes the infant from a sterile environment into a world teeming with bacteria and other infectious agents. Within a few days several of the common bacterial forms can be cultivated from his skin, conjunctivae, nasal passages, throat and intestinal tract. Ordinarily he arrives armed with natural defenses which are sufficient to keep these agents from invading beyond the surface of his skin or the mucous membranes of his intestinal tract. When an infection does occur, we postulate either that the infant's defenses have broken down or that he has been exposed to an agent of unusual virulence. To the persons in charge of newborn nurseries it is of considerable practical importance to understand which factor is primarily responsible. For, if the infant's defenses are at fault, the problem reduces itself to treatment of a single individual. But if an unusual agent is at fault measures must also be taken to prevent its spread to other infants. Sometimes the distinction can be readily made, but more often the presence of a circulating virulent agent is not discovered until secondary cases appear. The precautions which an individual nursery employs to shield its infants from epidemic infection vary according to the nursery's previous experience and the conditions under which its infants are delivered. At the Chicago Lying-in Hospital, probably due as much to good luck as good management, we have had no significant epidemic infections in the past 15 years and the small isolation nursery which we maintain stands empty most of the year. Consequently, I suspect our attitude toward some of the minor infections may be much more casual than that of Dr. De Pape who has suffered through epidemic infection of a nursery. I propose to consider some of the general ways in which we attempt to forestall infections and leave for Dr. De Pape a discussion of the clinical picture and management of severe infections.

Our concept of the infant's initial defense against infection is a complex of specific and non-specific factors derived in part from clinical experience and in part from laboratory studies. The part played by antibody passively transmitted from his mother, by specific and non-specific antibacterial properties of his blood, by relative tissue immunity and by gamma globulin, leucocytes and other factors varies for each infectious agent making it impossible to offer any useful generalizations. We know in a gross way that prematurity renders the infant more vulnerable to infection and that breast feeding probably makes

him a little more resistant. Thus, anything that can be done to prevent premature birth and to encourage breast feeding will probably be advantageous in controlling infections. In occasional instances we can ascribe infections to malformations, such as a ruptured myelomenigocele or omphalocele; or to hereditary defects such as agammaglobulinemia; or to the use of ACTH or cortisone. More often we are unable to understand exactly why an occasional infant admits the usual environmental bacteria into his tissues. Not a great deal can be done to anticipate these defects in the infant's defensive armor. In consequence, most of our efforts are directed toward decreasing the quantity of infectious agents around him with the main hope of excluding those virulent forms which constitute major hazards for him.

Prevention of infection begins during the prenatal care of the mother. Illinois state law requires that all pregnant women be screened for syphilis and appropriately treated if the disease is discovered. As a consequence, congenital syphilis has disappeared not only from our hospital, (which seldom delivers a woman who has not registered for prenatal care), but even in the state as a whole the incidence has fallen almost to zero. Our mothers are routinely subjected to microfilm chest examinations in a search for tuberculosis so that the infant may be isolated or immunized after birth if there is any question of active disease in the mother. Unfortunately our ability to screen fathers and collateral members of the family is not as well developed. Common respiratory infections in the mother at the time of labor are a potential danger to the infant, who should be separated from her at birth until the etiology of her infection is understood and she is no longer in a contagious stage.

A few infections may be transmitted to the infant via the mother's vaginal secretions during the period of labor and delivery. Our state law dictates that infants must be routinely treated with silver nitrate, whether or not there is evidence of gonorrheal vaginitis in the mother. Although we have chemical conjunctivitis, gonorrheal ophthalmia is unknown. When monilial vaginitis is identified before delivery the infant is usually

Presented at the Institute on Preventive and New Born Care, Manitoba Medical College, April, 1957. Dr. Wright's visit was made possible by a grant from the Borden Co.

treated prophylactically by swabbing the mouth with gentian violet at the time of delivery. Premature rupture of the membranes is a potential hazard, since retrograde infection of the amniotic fluid may lead to pneumonia in the infant even before birth. When membranes have been ruptured for more than 24 hours before delivery, antibiotics are usually administered to the mother and later to the infant until the absence of pulmonary infection seems clear. Our choice for the infant is a combination of penicillin and streptomycin. The same prophylaxis is used for infants who require resuscitation through a tracheal catheter. This measure is based upon the fear that the operator may transmit pathogens which he carries asymptomatically. Similar brief prophylaxis is used for small prematures and for any infant who is having trouble establishing normal respirations. Here the hope is that we can shield such infants from the additional jeopardy of a pneumonic component when they are already known to be suffering from atelectasis. It could be debated whether antibiotics used in this fashion are really effective and whether we are not promoting the emergence of resistant strains of staphylococci. Our justification lies in the fact that infants who come to autopsy almost never show histologic evidence of pneumonia unless it is clearly acquired in utero in an untreated mother. In addition we seldom use broad spectrum antibiotics as prophylactic agents, but reserve them for therapeutic indications.

From available statistics in the United States it seems clear that the trend away from home delivery of infants and toward hospital delivery is justified. Those geographic regions and classes of people which have the highest rates of hospital deliveries have the lowest maternal and infant mortality rates. However, it must be recognized that the concentration of mothers and babies into large units carries an increased hazard of spreading infection. Where the delivery unit consists of a physician, a nurse, and a small family, in the home, the number of potential disease vectors for the newborn infant is few. When, however, he is delivered in a large maternity hospital he may have direct or indirect contact with many nurses, physicians, attendants, necessary personnel and with other infants and their mothers. Unless constant vigilance is exercised a maternity hospital may serve as a vehicle for the dissemination of infectious agents. The types of control necessary must be suited to the individual hospital.

The Chicago Lying-in Hospital was constructed in pre-antibiotic days under the watchful eye of Dr. De Lee who had a horror of infectious disease. He not only insisted that the structure be physically separate from the rest of the University Clinic buildings but that no bridges or tunnels connect his maternity pavilion to the main portion of the clinics. Originally there was an entirely separate unit of the Lying-in Hospital for outpatients and for infected cases. In recent years this latter unit has been absorbed into the rest of the building, but it is still impossible for personnel or patients to pass from the main University Clinics to Lying-in without going out of doors. The brief exposure to outside air may not be an entirely sure method of killing infectious agents, but the inconvenience is a very effective means of cutting down the traffic between the two buildings, particularly in winter.

In addition to physical separation the lying-in Hospital has a staff which is almost completely independent of the general hospital staff. Neither graduate nor student nurses rotate to other parts of the University Clinics. The obstetricians infrequently consult outside of their own premises. The only real overlapping occurs through the resident and attending pediatricians who man the nurseries and at the same time have responsibility for patients in the children's hospital. The maternity hospital operates a separate laundry, formula room and kitchen for its patients and staff. How important a shield the physical and organizational isolation provides is difficult to evaluate, since the University Clinics as a whole have thus far escaped infections which spread in epidemic fashion. Those hospitals which are less fortunately arranged may, however, have to guard against the hazard of infection disseminated from contiguous units of a general hospital through common facilities and the intermingling of the staff.

The Chicago Lying-in Hospital as a unit enjoys another form of protection from infection by virtue of the fact that all of the infants cared for in its nurseries are born on the premises under a technique, which is as sterile as circumstances will permit. Nurseries, which must accept infants born outside under unsterile conditions, should admit them to a separate unit for observation until it is clear that they have escaped infection. Segregation of clean from unclean infants is particularly important in a premature nursery. Under the relatively ideal conditions described above we have in recent years been able to reduce our premature mortality to less than 10 per cent among infants between 2 and 5½ lbs. birth weight.

Because of his recognized susceptibility to infection the premature infant merits our most rigorous efforts at protection. In our unit this includes transportation from the delivery room in a closed, heated crib to an Isolette if he is under four pounds or is having respiratory trouble. The Isolette is housed in a constant temperature and constant humidity nursery from which he will not emerge until he reaches 5½ pounds. The personnel who minister to his needs are restricted to a minimum of nurses and physicians. Surgical scrub technique with soap containing hexachlorophene is used before these individuals enter the nursery and sterile gowns and masks are worn. Formulas are sterilized by the terminal method, and periodic spot cultures are taken to monitor the technique.

No contact of the infant with his mother or visitors is permitted until he reaches graduation weight. The most vulnerable aspect of these precautions is the health status of the personnel who work in the nursery. In theory it is logical to agree that anyone having the least suspicion of illness should feel morally bound to exclude himself from the nursery until fully recovered. In practice, however, it is inevitable that a conscientious nurse who knows that she will be replaced by an inexperienced substitute or none at all, will decide that the symptoms she feels are not related to infectious disease. Even if all members of the staff were entirely objective about their illnesses, there would still be no complete protection against the incubation stage of disease or the asymptomatic carrier state. The serious epidemics which have been reported in recent years are frequently traced back to such asymptomatic carriers. I have little confidence in periodic throat and stool cultures as a safety measure for recognizing carriers. In the first place they can provide no useful information unless repeated very frequently, and secondly the common bacteriologic techniques are insufficient to uncover some of the more dangerous agents. Unless a laboratory is prepared to undertake phage-typing of staphylococci and serologic grouping of colon bacilli and beta hemolytic streptococci, bacteriologic diagnosis will be inexact. Although these techniques are essential to the study of epidemics, they are far too cumbersome, expensive and slow for the ordinary day to day control of personnel. But in spite of the fact that our isolation techniques cannot be made absolute, premature nurseries everywhere have found that the measures outlined above provide adequate barriers to infection.

The care and housing of the full term infant who emerges from the delivery room is less arbitrary, since we know by experience that his risks from infection are smaller than the premature's and since we are unwilling to impose upon him separation from his mother. In the general nurseries, the staff is expected to abide by the same regulations of technique and health monitoring as with the prematures. However, we do not insist on masks. There is a more voluminous flow of personnel into the general nurseries, but those who have responsibilities therein are not simultaneously assigned to the care of adults. Greater use is made of attendants and student nurses. The infants are transported to their mother's rooms several times a day where they may have contact with other mothers but are shielded from visitors. Each of these concessions made for economic, teaching or psychologic reasons adds to the risk of the easy dissemination of infection to the unit of 20-30 infants, should we be so unfortunate as to have a virulent organism carried in. Nurseries which are able to arrange for small independent units or which have rooming-in are probably less vulnerable since an infection introduced will place only a small number of mother-infant combinations at risk.

One of the most difficult tasks which the conscientious medical staff of a nursery faces is the repeated necessity for judging whether aberrations from the normal are evidence of significant infection in a given infant. Since we have escaped serious trouble for many years, we are perhaps too casual in such judgments. The easiest way to administer anti-infectious measures is to banish any infant who shows the least suspicion into an isolation nursery. Our experience, however, has convinced us that a great deal of this concern is unnecessary and that we can afford to ride through many of the common aberrations without separating the infant from its mother and thus launching the mother-infant relationship on a note of anxiety. We seldom isolate infants because of thrush, purulent conjunctivitis, which we are sure is non-gonorrheal, paronychia, or minor infections of the umbilical stump. In addition we see superficial vesicular skin lesions which we have learned by experience will disappear promptly after the tops are wiped off and the base cleaned with alcohol. In years past we have even tried to pass such infections from one infant to another by diaper interchange, but without success. Most of the diarrhea we encounter is mild and appears to depend upon overenthusiastic feeding. If, however, it is not rapidly controlled by food restriction, or if the infant shows signs of fever, excessive weight loss or acidosis, we isolate him. I think it is quite possible that we are living in a fool's paradise, being sheltered in part by the special circumstances under which we receive our infants and in part by just good luck. To Dr. De Pape I shall leave a description of what happens when your luck runs out.

## Discussion

**A. J. DePape, M.D.**
Lecturer in Pediatrics, University of Manitoba
Pediatrician-in-Chief, St. Boniface Hospital

My first comment is one of appreciation to Professor Wright for his interesting talk on "Infections of the Newborn." I am sure that all those present will agree that this has been a most comprehensive and instructive period. Conversely, quite a few might disagree with Professor Wright when he states that the good results obtained at the Chicago Lying-in Hospital are due to good luck more than to good management; any nursery which has not had an epidemic infection in 15 years and which has a premature mortality, in the weight group of 2-5½ pounds, of less than 10% is exposed, in my opinion, to a very great deal of good management and a minimum of good luck.

Here in Winnipeg we have, in the recent past, had a series of epidemics due to staphylococcal infections in the nurseries. There was one in 1947 which persisted until 1949 and a second one in 1950 lasting to about 1951. In the course of these epidemics, practically without exception, the nurseries

in the city became involved in skin infections in the baby and breast abscesses in the mother at a rate running as high as 10-15 % of newborns and parturient women. This high morbidity resulted in a good deal of anxiety for parent, physician and hospital alike. It cast suspicion upon the hospital as the potential source of the infection, so that mothers feared delivery there; it discouraged breast feeding; it eventually spread to and caused subcutaneous abscesses amongst the attending staff, and finally it resulted in some neonatal mortality.

After much experiment with various antiseptic and antibiotic agents, the routine oral use of Aureomycin and later Chloromycetin was finally resorted to as a prophylactic measure for 4 days to all newborns, and this coincided with subsidence of the epidemic. Fortunately for us, we no longer are faced with a similar situation today.

Infections still occur in the nursery, however, and in the next few minutes I would like to review the role of infection in Perinatal Mortality as we have seen it at the Winnipeg General and St. Boniface Hospitals in the course of a Dominion-Provincial Public Health research project from April 1, 1954 to March 31, 1956.

Infection ranks, in our study, as the 4th commonest cause of Perinatal Mortality, with an incidence of approximately 10%.

**TABLE I**
**Perinatal Mortality***

April 1, 1954 to March 31, 1956 — (750 grams and 7 post natal days)

| | | |
|---|---|---|
| Anoxia | 78 | (24.1%) |
| Abnormal Pulmonary Ventilation and Diffusion | 52 | (16.0%) |
| Congenital Malformations | 50 | (15.4%) |
| Infection | 30 | ( 9.3%) |
| Hemolytic Disease of Newborn | 19 | ( 5.9%) |
| Trauma | 12 | ( 3.7%) |
| Miscellaneous | 11 | ( 3.4%) |
| Inconclusive | 9 | ( 2.8%) |
| Unknown | 42 | (13.0%) |
| Undiagnosed — No postmortem | 20 | ( 6.2%) |
| *Total WGH & SBH | 323 | (99.8%) |

It is seen more frequently in neonatal deaths than it is in stillbirths and more frequently in premature than in full term infants (Table II); indeed, it constitutes one of the major problems of premature care, a point thoroughly discussed by Professor Wright.

**TABLE II**
**Incidence of Infection in the Perinate**

| | |
|---|---|
| Stillbirths | 5.7% of 157 cases |
| Neonatal Deaths | *12.6% of 166 cases |
| Control Survivors— | |
| Premature | 14.7% of 102 cases |
| Full Term | 1.2% of 245 cases |
| Total | 7.1% of 670 cases |

*Slightly more than half of this 12.6% were prematures.

In the Perinatal deaths the lungs and placenta are more frequently involved, while in a fairly high percentage of cases the infection is generalized, the C.N.S. is also frequently involved.

**TABLE III**
**Sites of Infection**

| | Perinatal Deaths | Control Survivors |
|---|---|---|
| Lungs | 35 | 1 |
| Placenta | 12 | 1 |
| Generalized | 9 | --- |
| Central Nervous System | 6 | --- |
| Skin | 2 | 5 |
| Nasopharynx | 2 | 6 |
| Umbilicus | 2 | 3 |
| Heart | 1 | --- |
| Kidney | 1 | --- |
| Salivary Glands | --- | 3 |
| Intestines | --- | 7 |

In the control survivors the organs are somewhat different, the skin, nasopharynx and bowels being more frequently involved (Table III). This variation is, probably, technical in origin.

The coliform bacteria (Escherichia coli and Aerobacter aerogenes) and Pseudomonas aeruginosa constitute the most frequently occurring infective agents, but in this series at least, the staphylococcus (M. pyogenes var. aureus) is the commonest cause of Perinatal death.

**TABLE IV**

| | Perinatal Deaths | Control Survivors |
|---|---|---|
| M. Pyogenes— | | |
| Coagulase Positive | 15 | 5 |
| Coagulase Negative | 2 | 2 |
| E. Coli | 14 | 9 |
| Ps. Aeruginosa | 10 | 1 |
| Strep Fecalis | 2 | 1 |
| Aerobacter Aerogenes | 1 | 1 |
| Proteus Morganii | 1 | --- |
| Candida Albicans | --- | 1 |

About 10% then of our babies die of infection. It is our contention from the experience of the Perinatal Study, that if we are going to reduce Perinatal Mortality, the easiest way to do it would be by reducing deaths due to infection. To achieve this two things are essential:

1. Early diagnosis.
2. Early treatment.

**1. Early diagnosis of infection**

The diagnosis of infection in the newborn is almost never easy. Its presence will depend to a great deal on the nurse's ability to detect signs of disease and on the physician's awareness of the possibility of infection as the cause of this disease. Unfortunately, reliable specific signs are usually lacking in this age group or are confused with signs of other diseases. The newborn tends to show very few signs until terminally, when it is too late, and this is particularly true of the premature. There may be nothing more than a mild degree of listlessness, pallor or cyanosis; sometimes the baby merely feeds poorly or has a few loose stools; disturbances or respiratory or cardiac rate and rhythm are sometimes present; fever is most often absent, and the white cell count is normally high in the newborn. Almost all of these signs are variably seen in other common diseases of the newborn such as intracranial hemorrhage, pulmonary hemorrhage, hyaline membrane syndrome, etc. Indeed, many of them occur in the absence of organic disease, particularly in the premature.

As Professor Wright has pointed out, one is more suspicious of infection in any case where membranes have been ruptured 24 hours, where resuscitation and tracheal intubation have been necessary, where there is evidence of respiratory distress and in any premature weighing less than 3½ lbs. We would add all cases of explosive diarrhoea and those cases of jaundice where the liver is enlarged and the umbilicus red, possibly with or without purulent discharge. Here chest films and early cultures of the throat, blood, stool, urine and C.S.F. are often of value.

2. **Early treatment of infection**

There can be no doubt that the best management of infection in the newborn is prevention.

(a) Prevention in not allowing infection into the nursery.

(b) Prevention in the use of prophylactic antibiotic therapy.

The nurse's role in the prevention of transmission of infection to the newborn and particularly the premature is paramount; she is in more intimate and more constant contact with the baby than anyone else and any carelessness on her part in the use of masks and gowns, and particularly the careful scrubbing of the hands (preferably with a hexachlorophene soap) in between the handling of each patient can result in disaster .

Where the diagnosis of infection is established or strongly suspected, the infant should be immediately transferred to the isolation nursery, and a suitable antibiotic in adequate dosage prescribed. There is some controversy as to the choice of antibiotic for both prophylactic and therapeutic use. Professor Wright has suggested the use of Penicillin and Streptomycin for prophylaxis and the broad spectrum antibiotics for therapeutic use. We would agree with him emphatically about the latter, but our experience with Penicillin and Streptomycin has not been too satisfactory even for prophylaxis and some of us use the broad spectrum antibiotics for this as well.

For active therapy Chloromycetin in doses of 35-75 mgms. per kilo per day has found favor with some, while others prefer one of the Tetracycline derivatives such as Terramycin in doses of 25-50 mgms. per kilo per day. Sometimes Polymyxin and Neomycin may have to be used as well. In some cases however, and no matter what the choice of the antibiotic, the result is still too often fatal. This may be due to important differences of immunity, particularly in the premature, or it may be due to non-susceptible agents such as viruses.

To summarize then the best management of infection consists of preventing it from getting into the nursery, of isolating any case of infection immediately and instituting antibiotic therapy.

Lastly, and probably most important of all, with respect to diagnosis, one should suspect infection in any baby who is ill and there is no other reason for the illness.

## Abstracts from the Literature

**The Treatment of Cardiac Arrest Occurring During Surgery.** Kay, J. H., Dever, R., Gaertner, R. A., and Kaiser, G. C.: J.A.M.A.; 163: Jan. 19th, 1957, 165.

The authors describe a simple and effective routine for the treatment of cardiac arrest based on their experience with over 300 experiments on cardiac arrest in dogs and on the cases of 10 patients with ventricular fibrillation and 20 with ventricular standstill who were treated successfully.

Cardiac Arrest occurs in two forms: ventricular standstill and ventricular fibrillation. The anaesthetist usually discovers that cardiac arrest has occurred by his inability to detect any blood pressure or carotid artery pulsation. If the surgeon is working in the abdomen or chest he can verify the diagnosis by palpating the aorta or heart. If he is away from the large vessels he can incise the skin or enlarge his incision, and if no bleeding ensues, then cardiac arrest has occurred.

Treatment consists of immediate thorocotomy through the left 4th interspace. The heart is palpated and massage is begun.

**General Principles**

(a) Artificial respiration by any means available, i.e., mouth-to-mouth insufflation, anaesthetic bag and mask or bag and endotracheal tube.
(b) Determine whether ventricular fibrillation or ventricular stand-still has occurred.
(c) Massage first until heart is firm and pink; then inject the chosen drug or defibrillate.
(d) Inject drugs into the left ventricle.

**Specific Treatment**

1. **Ventricular standstill**
   (a) Epinephrine 1 cc. of 1: 1,000 diluted to 10 ccs. with normal saline—2-3 ccs.; if ineffective this may be repeated many times.
   (b) Calcium Chloride 2-4 ccs.
2. **Ventricular fibrillation**
   (a) Electrical defibrillation
   130 volts for 0.25 secs. or
   220 volts for 0.10 secs. or
   a series of shocks at 0.5 secs. intervals up to 6 or 8.

M. Minuck, M.D.

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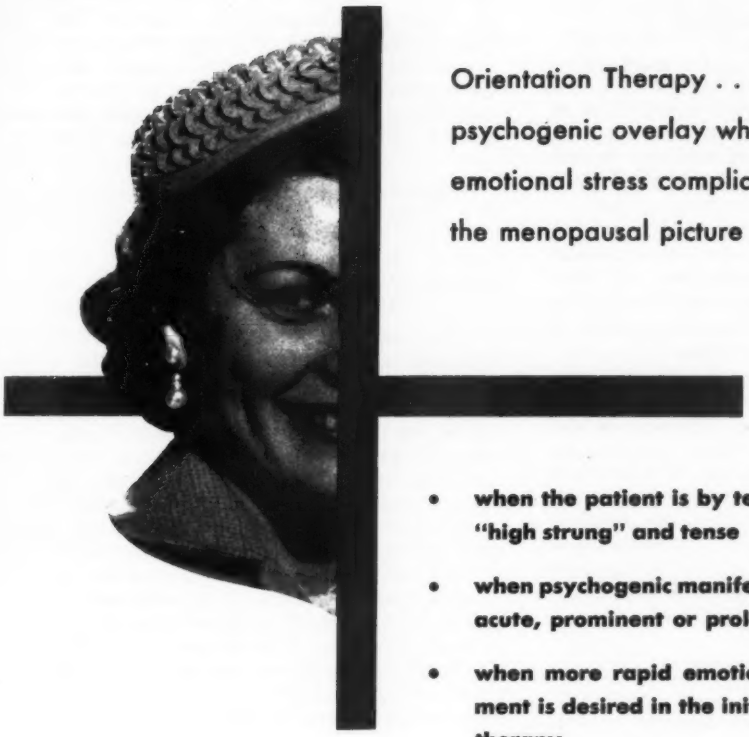
91 S



Orientation Therapy . . . to remove the psychogenic overlay when unusual emotional stress complicates the menopausal picture

- **when the patient is by temperament "high strung" and tense**
- **when psychogenic manifestations are acute, prominent or prolonged**
- **when more rapid emotional adjustment is desired in the initial stage of therapy**

# "PREMARIN" with MEPROBAMATE

Ayerst

*To remove the psychogenic overlay . . .*
Meprobamate reduces tension, lessens irritability and restlessness, promotes more restful sleep and generalized muscle relaxation.
(Borrus, J. C. P.: J.A.M.A. *157*:1596 (Apr. 30) 1955.)

*To treat the basic estrogen deficiency . . .*
"Premarin" supplements declining endogenous estrogen levels and provides prompt symptomatic relief of distressing symptoms plus a gratifying "sense of well being."

***Ayerst, McKenna & Harrison Ltd., Montreal***

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# Biochemistry

## Serum Transaminase

F. D. White, M.D.
Professor of Biochemistry, University of Manitoba; Biochemist, Winnipeg General Hospital

The estimation of the transaminase content of the serum is a procedure which has recently been introduced into the schedule of analyses carried out in the Biochemistry Department of the Winnipeg General Hospital.

This test, developed during the last couple of years, has proved to be of considerable diagnostic significance in myocardial infarction and liver necrosis, and its increasing importance suggested that a brief sketch of the biochemical background to the test might be of some interest.

Transaminase is an enzyme which plays an important part in protein metabolism. When the protein of the diet undergoes digestion in the gastro-intestinal tract, it is broken up into its component amino acids, which are then absorbed into the blood stream, and thus transported to the liver and other tissues where they are metabolized. The first step in amino acid metabolism is almost invariably deamination, an enzymic process whereby the amino acid is converted to the corresponding keto acid with elimination of ammonia.

e.g.

$$R{\cdot}CH(NH_2)COOH \xrightarrow{-(H_2O)} R{\cdot}CO{\cdot}COOH + NH_3$$

(amino acid) (keto acid) (ammonia)

Now this reaction results in a continuous production of ammonia, a toxic substance, which therefore has to be utilized or eliminated as soon as it is formed. This process proceeds along three main pathways.

The first of these, which accounts for most of the ammonia, involves its conversion to the innocuous and easily excretable urea; by the second, ammonia combines with glutamic acid in the liver to form glutamine, which is then carried by the blood stream to the kidney, where an enzyme, glutaminase, splits off the ammonia again for immediate excretion as urinary ammonia; the third pathway is transamination. It is now generally accepted that protoplasm exists in a state of flux, being constantly broken down and resynthesized. For the resynthesis of protein, the tissues draw upon a common pool of amino acids and their keto equivalents, but different tissues have different proteins and consequently different amino acid requirements. One tissue might therefore have at its disposal a plethora of one amino acid and not sufficient of another, were it not for transamination, a process which gives fluidity to the composition of the amino acid pool. This is carried out by means of enzymes, the transaminases, which have the ability to remove the amino group from amino acid "A" and transfer it directly to the keto group of acid "B," thereby producing amino acid "B" and thus increasing the content of the latter at the expense of amino acid "A." The most important and best characterized of these enzymes is the 1-glutamic acid transaminase, which has as its co-enzyme, pyridoxal phosphate (vitamin $B_6$), a substance which acts as the carrier of the $NH_2$ group from the amino acid to the keto acid.

Glutamic acid (amino acid) + Oxalacetic acid (keto acid)

↓ ↑

$\alpha$ keto glutaric acid (keto acid) + Aspartic acid (amino acid)

This reaction proceeds continuously within the cell, but when the cell is destroyed, the enzyme passes out into the blood stream, thereby increasing the enzyme content of the serum. To determine the amount of increase it is only necessary to take a measured amount of serum and incubate it with excess of aspartic acid and keto glutaric acid, and the greater than normal amount of glutamic and oxalacetic acids formed is a measure of the increase in serum transaminase.

Clinical trials have shown that a rise in serum transaminase may be expected within 24 hours in cases of myocardial infarction and that this will subside to normal 3 to 5 days later. With liver necrosis, the peak is generally much higher, and is slower in coming back to normal. For details, see the accompanying paper by Dr. Hugh Ross.

## Survey of 67 Serum Glutamic-Oxalacetic Transaminase Determinations (SGO-T)

Hugh M. Ross, M.D.
Department of Pathology, Winnipeg General Hospital

Professor White has reviewed the significance and biophysiology of the group of enzymes that are the subject of our discussion. More specifically we are interested in one of these, the serum content of glutamic-oxalacetic transaminase.

The introduction of a new procedure into a busy department already operating far in excess of its present facilities presented considerable problems. The routine procedure must be capable of being easily and conveniently performed by already overtaxed technicians. It must have sufficient accuracy and reduplicability to be of value to the clinician. Consequently, we explored a number of procedures the details of which I will not dwell upon at the present time. Miss Catherine Gibson, Dr. Peter Mierau, and Dr. Bohdan Wolanskyj all participated in this, and Dr. White and myself are merely reporting the results of our combined efforts.

Since the object of this was to provide as quickly as possible the clinical staff of the hospital with a procedure that they and the Department of

Presented at a Clinical Luncheon, Winnipeg General Hospital, February 21, 1957.

Biochemistry agreed was necessary, it was not possible to carry out extensive preparatory investigations into its use. We found, as previously reported, that the normal range of SGO-T was between 10 and 40 units with a mean of about 20 units.

I thought, however, it would be of interest to see how this procedure which became available about the first of the year, was utilized, and to determine its value, if any, in the treatment of our patients. I was impressed generally with how well it was requested. Only in a few cases, in my appraisal of the charts, did I feel that the test had been one of the pellets of a diagnostic barrage loaded without any particular care and aimed haphazardly in the vague direction of a diagnosis.

I have attempted to tabulate the tests performed as to their purpose and to consider how they helped the clinician to better assess and treat his patient.

I investigated 67 examinations, performed on 37 patients, over about a 6 weeks period beginning with the time when the test became available as a routine procedure. In the great majority of cases the test was used to confirm the clinical impression that the chest pains of the patient were either of myocardial ischemic nature, without necrosis or were unrelated to the cardiovascular system. There were 17 such cases in which the normal SGO-T estimation supported the diagnosis, many of which were on multiple examinations. It would seem that the test was of considerable value in assessing these patients, some of whom were reported as showing some atypical electrocardiographic changes.

In 4 cases, the elevated SGO-T levels and typical pattern confirmed the presence of a myocardial infarct also demonstrated unequivocally by clinical observations and electrocardiographic changes.

In 5 cases, the presence of myocardial infarction was definitely established ultimately, but the serum transaminase was elevated before the typical electrocardiographic changes were evident. This was demonstrated by the following case:

**Case 1**

Mr. T. B., a 57 year old man who had a sudden onset of severe precordial pain at 4 a.m. the morning of January 13, 1957. The first ECG was reported as borderline normal and although the repeat record was abnormal, infarction was not diagnosed. The SGO-T on the 14th was 157 units, but two days later was within normal limits. By the 21st the ESR was elevated to 30 mm. and the electrocardiogram suggested an antero-lateral infarction, which showed the characteristic resolvement pattern. The patient was put on anticoagulant therapy from the first and treated as an infarction and made an uneventful recovery.

Two cases were symptomless and the SGO-T were normal. There were 3 cases where there was fairly conclusive evidence of infarction, yet the SGO-T levels were not elevated. In two of these transaminase levels were taken six days after the onset of infarction, and would be expected to be normal.

**Case 2**

Mr. H. T., an 82 year old man with angina pectoris had a sudden onset of unrelenting precordial pain at 5 p.m. on January 3, 1957. The ECG, though abnormal, did not show signs of an acute infarction until the January 5th tracing which was followed by characteristic resolvement. Though the ESR was always normal, the WBC was elevated on the 9th. SGO-T levels on the 3rd, 7th and 8th were never elevated. It is possible that the peak of a small infarct was missed.

In two cases, the SGO-T levels gave evidence of an extension, or occurrence of a second acute infarction.

**Case 3**

Mrs. R.P., a 43 year old diabetic admitted in impending coma and precordial pain on January 14th. The ECG showed diffuse ischemic changes and again on the 17th. However, the SGO-T level was elevated to 63 units on the 14th. Two subsequent levels were negative. On the 22nd, recurrence of pain was accompanied by an elevated SGO-T level to 59 units and the ECG showed an anterior acute infarction which subsequently resolved.

**Case 4**

Mrs. R. L., a 80 year old woman with a long history of congestive heart failure successfully controlled by digitalis and diuretics variously felt to be due to rheumatic valvular disease and to arteriosclerotic heart diseases. She was seized with severe, unradiating precordial pain at 1.40 p.m. on February 12, 1957 and was fibrillating and cyanosed on admission. ECG showed no evidence of recent infarction. Her SGO-T level was the highest in the series — 420 units. She developed Cheyne-Stokes respirations and died. At autopsy, she had extensive mitral and aortic stenosis with a soft mural thrombus in the left auricle and extensive antero-basal recent infarction. We feel that this is one of the rare embolic cases. There was little coronary arteriosclerosis.

Only 5 cases had transaminase levels in an assessment of liver function. In one the presence of proved liver metastases failed to show an elevated SGO-T.

I have presented a survey of the use of serum glutamic - oxalacetic transaminase determinations in its inception in this hospital. Naturally, conclusions are difficult with the small numbers involved. It would seem that the procedure is of considerable value in ruling out myocardial infarction in cases of suspected myocardial ischemia. In a few cases, the procedure was an earlier evidence of infarction than the ECG. The correlation in the use generally, was remarkably good.

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## Central Cancer Registry

In February 1956 the Central Cancer Registry was re-organized. To most practitioners the event was marked only by the introduction of a new cancer report form, a questionnaire even more detailed than the last, to be completed by him and returned to the Department of Health.

The act of making cancer a reportable disease in 1928 marked the first interest of the provincial government in the cancer problem. In 1930 the Government created the Manitoba Cancer Institute "to take such steps as may be considered advisable by the board for the relief or cure of cancer in the Province of Manitoba."

In order to evaluate its program, the Cancer Institute started a statistical service which initially was based upon the registered death rates. It was found that, while cancer was a reportable disease, the reporting was not sufficiently comprehensive to permit statistical analyses. As a consequence of the ever increasing interest in cancer control, the reporting steadily improved until by 1942 it was felt that there was sufficient data to be of some statistical value. It was at that time that the Provincial Government turned over to the Cancer Institute all the records on cancer reports from the year 1928.

In 1944 the Institute provided a secretary to the Winnipeg General Hospital's Tumor Service, and this materially improved the cancer reporting. In 1944 it established a Cancer Follow-up Service in the Winnipeg General Hospital, whereby an attempt was made to register all patients treated for cancer who passed through that hospital, at the request of either the physician or the hospital, and to take such steps as were necessary to assist in having patients returned for follow-up examinations. A similar service was established in St. Boniface Hospital in 1951. These steps greatly expanded the effectiveness of cancer reporting.

With the advent of the Cancer Health Grants in 1948 the Institute was enabled to expand its statistical studies. In 1950 the National Cancer Institute appointed a Statistical Officer, and in 1952 a full-time statistician was appointed.

In February 1956 a new "cancer report" form was devised containing what was considered to be minimum necessary detail on each new cancer case. These forms are returned to the Department of Health and are then sent to Central Registry, where the information they contain is coded and transferred to "abstract cards" from which it may easily be recorded on the I.B.M. punch card. Thus, information from every malignant case occurring in the province is collected and coded at Central Registry. A fairly large number, of course, (about 17%) are first notified at death by the Division of Vital Statistics. The information obtained on these cases is also recorded on punch cards. These cards are then filed at Central Registry and are reviewed each year on the anniversary of the first treatment, at which time a report from the doctor who originally saw the case is requested and this follow-up report is put on the punch card.

The information being collected will be of the greatest value in the future diagnosis and treatment in our Province. It will make possible the evaluation of the efficacy of any general type of treatment, it will facilitate cancer incidence studies and will provide a source of information which will be available to all practitioners in this province making a study of any particular aspect of the cancer problem.

When the Cancer Incidence Study, sponsored by the National Cancer Institute, gets underway in August we will be able, with little modification, to make use of the existing system which is already functioning.

It is hoped in future to publish from time to time in these columns the information gleaned from Central Registry statistics which will be of interest to Manitoba practitioners.

D. W. Penner, Chairman,
Cancer Incidence Study Committee.

## A Cancer Incidence Study for Manitoba

In the preceding article the history and function of the Central Cancer Registry has been reviewed. That data is being recorded on cancer patients is well known to most Doctors in Manitoba. For many years they have been faithfully providing the data on their patients which has made it possible to collect information on cancer patients

which is second to none in Canada. Much credit is due the profession for this praiseworthy achievement. The National Cancer Institute has now proposed a nation-wide Cancer Incidence study under its' sponsorship. The first province to participate was Newfoundland, which commenced its study in June, 1956.

Cancer incidence is a measure of the cancer problem in a population and is the number of new cases which arise in any specific period of time, generally one year. Such information is much more meaningful than cancer mortality since it indicates the magnitude of the cancer treatment programme and the extent of the patient follow-up programme.

In Manitoba a committee has been formed to guide the Cancer Incidence Study in this province. Composed of representatives of the various groups interested in the cancer control programme, the membership of this committee is as follows:

Dr. D. W. Penner (chairman), representing the Canadian Association of Pathologists.

Dr. J. E. Hudson, President M.M.A.

Dr. J. A. Findlay, Brandon, representing M.M.A.

Dr. C. W. Hall, representing Radiological section of M.M.A.

Dr. L. O. Bradley, representing the Greater Wpg. Regional Hospital Council.

Dr. R. J. Walton, representing the Manitoba Cancer Institute.

Mr. R. Hooper, representing Manitoba Division Canadian Cancer Society.

Because of the existing program already in operation in Manitoba, co-operation with the National Cancer Institute of Canada is relatively simple.

The statistician of the National Cancer Institute of Canada, Dr. A. J. Phillips, will assist the analysis of the data, and already Dr. Phillips has met with the committee to organize the study. It is understood that the results of the study will be released only with the approval of the provincial cancer incidence committee.

The success of this project is largely dependent upon the continued active co-operation of the medical profession in the province. The committee is hopeful that every doctor in Manitoba will ensure that each new cancer case is reported as soon as the diagnosis is made. Letters of explanation regarding the procedures to be followed are presently being distributed to all doctors in the province. We earnestly request your co-operation and assistance.

D. W. Penner, Chairman,
Cancer Incidence Study Committee.

# ANNUAL CONVENTION

## Manitoba Medical Association

(Canadian Medical Association, Manitoba Division)

## Winnipeg, October 15, 16, 17, 18

## Tentative Program

**Scientific and Business Sessions will be held in the**

**Royal Alexandra Hotel**

### Guest Speakers

Dr. H. B. Atlee, Head of Department of Obstetrics and Gynecology, Dalhousie University, Halifax.

Dr. J. W. Gerrard, Professor of Paediatrics, University of Saskatchewan, Saskatoon.

Dr. J. A. L. Gilbert, Medical Arts Building, Edmonton, Alberta.

Dr. R. C. Harrison, Asst. Prof. Clinical Research, University of Alberta, Edmonton, Alberta.

Dr. M. A. R. Young, President, Canadian Medical Association, Lamont, Alberta.

Mr. L. W. Holmes, Assistant Secretary, Public Relations, Canadian Medical Association, Toronto.

### Tuesday, October 15th

**Morning**

9.30 Medical Health Officers, Section of the Manitoba Public Health Association.

**Afternoon**

2.00 Executive Committee.

5.00 Manitoba Medical Association.

**Evening**

6.00 President's Dinner to Retiring Executive.

8.15 Annual Meeting Business Session—Members only.

Report of the Nominating Committee.

Presidential Address.

### Wednesday, October 16th

**Crystal Ballroom**

**Morning**

8.15 Registration.

8.30 Film.

8.55 Opening of Scientific Session, Dr. J. E. Hudson, President.

9.00 Chairman — Acne and Acne Scars, Dr. S. Berger.

9.30 Gall Bladder Disease, Dr. A. C. Abbott.

10.00 New Wine in Old Jugs, Dr. S. Vaisrub.

10.30 Intermission: Visit the Scientific and Technical Exhibits.

11.00 Chairman — Fashions in the Treatment of Carcinoma of the Breast, Dr. R. C. Harrison.

**Noon**

12.00 Luncheon.

Guest Speaker: Dr. Morley A. R. Young, President, Canadian Medical Association.

**Afternoon**

2.00 Chairman — The Use of Sulphonylurea Compounds in Diabetes, Dr. J. A. L. Gilbert.

3.00 Laboratory Session
Virus
Dr. J. C. Wilt
} Panel

Other speakers and topics to be announced.

**Evening**

6.30 Sectional Meetings.

8.30 Annual Meeting Business Session, Continued

## Thursday, October 17th

### Crystal Ballroom

**Morning**

8.30 Registration.

8.30 Film.

9.00 Chairman —
Treatment of Chronic Ulcerative Colitis,
Dr. A. G. Rogers.

9.30 Multiple Injuries, Assessment and Management of,
Dr. J. S. McGoey.

10.00 Oesophageal Hiatus Hernia,
Dr. C. M. Burns.

10.30 Intermission: Visit the Scientific and Technical Exhibits.

11.00 Chairman —
Pre-diabetic Syndromes,
Dr. J. A. L. Gilbert.

**Noon**

12.00 Luncheon.
Guest Speaker: Dr. H. B. Atlee,
Cancer Pelvic Organs and the G.P.

**Afternoon**

2.00 Chairman —
Limitations in the Surgical Treatment of Duodenal Ulcer,
Dr. R. C. Harrison.

3.00 Annual Meeting Business Session
Continued.

**Evening**

Annual Dinner and Dance

6.30 Reception.

7.15 Dinner

9.00 Dance.

## Friday, October 18th

### Colonial Room

**Morning**

8.30 Registration.

8.30 Film —

9.00 Chairman —
Moles,
Dr. W. A. Maclean.

9.30 Recent British Practice in Obstetrics and Gynaecology,
Dr. Jean McFarlane.

10.00 Impending Myocardial Infarction —
Recognition and Treatment,
Dr. R. E. Beamish.

10.30 Intermission: Visit the Scientific and Technical Exhibits.

11.00 Chairman —
Dr. H. B. Atlee.
First Ten Minutes of a Baby,

**Noon**

12.00 Luncheon.

**Afternoon**

2.00 Care of the Unborn Child,
Dr. J. W. Gerrard.

3.00 Paediatric Symposium.
Dr. H. Medovy, Moderator,
Including Dr. Fischel Coodin:
Acute Diarrhoeas.
Dr. J. W. Gerrard, "Chronic Diarrhoeal Disorders in Infants and Children.

# Editorial

S. Valsrub, M.D., M.R.C.P. (Lond.), F.R.C.P. (C.), F.A.C.P., Editor

## Pain

The painstaking physician dutifully questioning the patient about the location, radiation, character and duration of his pain, views pain as a clue to diagnosis. Having in due course established a diagnosis, no longer the sleuth, the physician now begins to regard pain as a symptom to be alleviated. The patient, of course, views pain less dispassionately. To him it is a most unwelcome guest to be rid of with the greatest possible despatch.

To the above points of view within the context of patient-doctor relationship may be added those of the psychiatrists, who see in pain a frequent substitute for neurosis, a possible refuge from an intolerable situation, an attempt at winning sympathy, or even an expression of suppressed aggression. They find that whatever its cause, pain may in time become the patient's main interest and pre-occupation, a prop essential to his existence. Twenty-six of the two hundred and seventy-five patients operated on by John Penman (The Lancet; 1, 633, March 1954) for Tic Doloreux, have lapsed into apathy or depression upon their release from pain. Not unlike the Prisoner of Chillon, the patients found no joy in their newly found freedom.

It should not be inferred, however, that the variety of views on pain is confined to the sphere of Medicine. Pain is the concern of every human being. It is a favorite topic for discussion in all walks of life. It is conspicuous in the works of philosophers, theologians, scientists, writers and artists of all lands and all races. As early as the 4th century B.C. Aristippus of Cyrene based his philosophy on avoidance of pain. Postulating that the moment that is not filled with pleasure will let in pain, he advocated the pursuit of pleasure as the only way of life. Similarly, Epicurus regarded pain as the sole evil and the absence of pain as the highest good. Seneca, the Stoic, considered pain to be an act of judgment, something to be ignored and dismissed from the general scheme of things. Religious philosophers viewed pain as an act of punishment, redemption, or a trial of virtue, something interwoven closely with the concept of sin and repentance. "Scientific" philosophers of modern vintage tended to regard pain as a teleological device, a warning of impending danger, which protects the species and enables it to survive. Writers, poets, musicians, painters, sculptors found in pain a source of inspiration as well as material for their creative works.

The variety of approaches to pain bears testimony to the dominant position of the latter in the minds of men. It also suggests that not all the approaches lead to the same objective. It may well be that not all who speak of pain refer to the same concept. Pain may have more than one meaning.

What is pain? An answer that comes readily to mind, one that can hardly qualify as a definition, is, that pain is the antithesis of pleasure. In this broad and vague sense pain is more akin to emotion than sensation, more symbolic than existential. Painful duties, painful memories, painful tasks and similar expressions illustrate this concept, which equates pain with suffering. This is a popular, widely accepted connotation of pain.

In a narrower sense pain is a perception—a specific sensation with specific subservient physico-chemical mechanisms. This concept, now taken for granted by every physician as well as every layman with some knowledge of neuroanatomy, is relatively new. The ancients had no such conception. Even Aristotle, so thorough in his classification of the special senses, failed to see in pain a separate specific perception. He regarded it as an unpleasant quality in the sense of touch. A logical outgrowth of this view was the "intensive" theory, widely held as late as the nineteenth century, according to which pain was the result of intense stimulation of any of the special senses. The first step in the separation of pain from other sensations was taken by Weber in 1846, when he placed pain in the category of common sensibility along with such vague sensations as nausea and vertigo. It was not, however, until the end of the nineteenth century that pain was elevated by Max Von Prey to the rank of a special sensation with a special neural apparatus.

Subsequent researches have amplified knowledge and understanding of the physical mechanism of pain. The pathways of conduction of pain in the peripheral nerves, dorsal roots and spinothalamic tracts, have been clearly demonstrated; the role of the thalamus in the awareness of pain, that of the cerebral cortex in the finer discrimination of its quality and intensity, and the part played by the short intercalary neurones in its modification and augmentation, have been elucidated. The differences between the various types of pain—somatic, visceral, thalamic, localized, referred—have been clarified. Many other aspects of the physical basis of pain have been explored.

The most recent contribution of significance to the understanding of the psycho-physics of pain is that made by Hardy, Wolfe and Goodell, who have devised an apparatus and a technique for the study of pain, the pain threshold, and the pain threshold raising analgesics. Their apparatus, known as the dolorimeter, determines the pain threshold by focussing radiant heat through a solenoid shutter onto the skin. The pain threshold

is measured in millicalories of heat, generated by a 500 watt projection bulb, per second per square centimeter of exposed skin, required to elicit a distinct stab of pain at the end of three second exposure. The results of these investigations published by Hardy, Wolfe and Goodell in numerous papers and in their book "Pain Sensations and Reactions" (Williams and Williams) show a high degree of uniformity of the pain threshold and a considerable usefulness of their technique in evaluation of analgesics. While these results have not gone unchallenged, and much doubt has been cast by many observers on the uniformity of the pain threshold and on the value of the dolorimeter in the evaluation of analgesic drugs, it is generally conceded that the work of Wolfe, Hardy, and Goodell constitutes a most significant recent advance in the study of the physiology of pain.

Having quantitated pain, Wolfe, Hardy and Goodell were careful to point out that all they were measuring was the perception of pain, not the complete pain experience, which is a composite of pain perception and pain reaction. The latter is a complex of associated sensations, memories, anticipations, attitudes, hopes, ideas and emotions, and does not lend itself too readily to measurement and controlled experiment. It is this emotional aspect of pain which gives it the added quality of symbolic significance.

The emotional component of pain is a factor of great importance in the evaluation of any method of its treatment. This is attested to by the success of placebos, and by the painstaking care to ensure the double blind method in drug evaluation. The reaction to pain also renders its treatment more complex and varied. This is apparent not only in the pharmacotherapy of pain, but also in the surgical methods of treatment, (Neurosurgical Procedures for the Relief of Intractable Pain, by Harold N. Lynge and Gerd Fischer, in this issue), among which one finds alongside a simple nerve section directed at the ablation of pain perception, the operation of prefrontal lobotomy aimed at the modification of the emotional reaction to pain.

Pain, thus, emerges as a complex experience—a perception, a feeling state, a symbol. Despite the light thrown upon it by the lamp of science, it is but poorly understood, for it is a part of a yet greater mystery, that of the relationship of body and mind. To quote Strauss, E. B. (Brit. Med. J., 11, 411, 1949): "It is impossible to embark on a discussion on pain without being brought up with a bump against the insoluble body-mind problem. All experience, of course, has its origin in sensory data. Sensation becomes perception; percepts are further elaborated into concepts; concepts cluster together to form ideas. . . it is a habit of mind to think of pain in physical terms and to draw an artificial distinction between real and imaginary pain. All pain in final analysis is a psychic event."

The pain experience, embracing as it does perception, emotion, memory and anticipation is so ubiquitous and all-pervading, that it is difficult to visualize life without it. Yet, curiously enough, there are cases on record of individuals who have never felt pain. (Congenital Indifference to Pain, Macdonald Critchley. Ann. Int. Med. 45: 5, 757, 1956). Interestingly, these people managed to survive, albeit not unscathed by trauma, despite this "handicap." Is it possible that the warning bark of the watchdog has been somewhat overrated?

Whatever its cause, its mechanism and its purpose, pain is a cruel and tyrannical master. Freedom from pain, unmentioned in political declarations will never be omitted from a Medical Manifesto.

—Ed.

## College of General Practice of Canada

**Manitoba Chapter**

### Notice of Post Graduate Course

There will be a post-graduate course of 20 lectures in Medicine for practising doctors, commencing September 18th, 1957. These will be evening lectures — once weekly — starting each Wednesday evening at 8.15 p.m.

The Speaker: Dr. Sidney Israels.

The Subject: Applied Principles of Physiology and Biochemistry to the diagnosis and treatment of diseases in general practice.

The Place: Theatre A — Manitoba Medical College.

Attendance will be allowed for creditation of members in the College of General Practice of Canada.

All doctors are invited to attend.

## College of General Practice of Canada

**Manitoba Chapter**

### Notice of Annual Meeting

The Annual Meeting of the Manitoba Chapter of the College of General Practice will be held in the Chalet at Clear Lake, Manitoba, September 7th and 8th, 1957.

There will be a Scientific Session, Annual Reports, Election of Officers, and Entertainment.

All medical doctors are invited to attend.

## Letters to the Editor

Dear Editor:

May I extend through your journal the thanks of the Medical Library Committee, the Dean, Miss Monk and myself for the wide-spread response to my letter regarding the retirement of our Librarian? Donations were received from all parts of the province, many with little notes of appreciation which, I believe, meant much to Miss Monk.

In spite of the heat, some fifty persons attended the presentation in the Medical Library on July 17th. Dean Bell pointed out that the present satisfactory state of the library was a monument to Miss Monk's thirty-six and a half years of steady search for improvement in service to student and practitioner alike. He presented her with an engraved wrist-watch as a token of the high esteem in which she is held. President Saunderson offered the best wishes of the University as he presented her with a substantial purse. Miss Monk's reply drew applause from all.

While the appeal did require some extra work and time, the result has again demonstrated the profession's warm gratitude to those whose service leads to advances in training and practise.

Sincerely,
M. J. Ormerod, M.B., Chairman,
Medical Library Committee.

Dear Editor:

May I please have a little space in the Review to say "Thank You" to the doctors of this province and to the medical faculty for the beautiful wrist-watch and the substantial cheque that was presented to me on the 17th of July.

For these two splendid gifts I thank you very much indeed, but what touches me beyond words is the thought that prompted their giving.

The years since 1921, when I joined the medical library staff, have been extremely happy ones for me. There is much more that I wish that I could have done for you, but the life-time of one individual is very little in the history and the developing of an institution.

In saying "Farewell" to you, I will repeat what I said on the 17th—"From here, like "Don Quioxte" I shall move on, seeking ever the new and adventure, and at the journey's end you will find engraven upon my heart, the name of this Medical Library.

Faithfully yours,
Ruth D. Monk,
Medical Librarian.

## Obituaries

### Dr. Laurance Thornton Ainley

Dr. Laurance Thornton Ainley, former senior pension officer of the Dept. of Veterans Affairs died May 19 aged 79. Born at Chamblay, P.Q., he graduated in Arts (1900) and Medicine (1904) from McGill University. He practised at Wadena, Sask., served overseas in the first world war and on his return was appointed pension officer with the D.V.A., retiring in 1948.

♦

### Dr. Everett Joseph Washington

Dr. Everett Joseph Washington, 74, died on May 23. Born in Portage la Prairie, he grew up at Ninga where his father was a prominent farmer and Shorthorn breeder. He attended Wesley College and Manitoba Medical College, graduating with the M.D. degree in 1909. Later he did postgraduate work in Chicago, then returned to Winnipeg where he practised until his death.

He was professor of otolaryngology in the Faculty of Medicine, University of Manitoba, past president of the Canadian Society of Ophthalmology, a Fellow of the Royal College of Surgeons of Canada, and a life member of the Winnipeg Medical Society.

Surviving him are his wife, two daughters and a son and seven grandchildren.

♦

### Dr. Abraham Hollenberg

Dr. Abraham Hollenberg, 57, died suddenly June 16. Born in Austria, he was educated in Winnipeg schools and the University of Manitoba (B.A. 1920, M.D., B.Sc. 1924). In 1926 he was appointed demonstrator in medicine and rose to be Associated Professor in 1951. Twice he was honorary president of the Manitoba Medical Students Association.

He was a member of the Board of Trustees and of the Executive Committee of Manitoba Medical Service. From 1943 to 1948 he served as Winnipeg member-at-large of the Manitoba division of the Canadian Medical Association. He was vice-president of the Shaarey Zedek Synagogue congregation.

He is survived by his widow, a daughter Dr. Barbara Kaufman, Cleveland, O., two sons Dr. Charles H. and Martin; four brothers Drs. Michael, Joseph, Charles and Jacob, two sisters and one grandchild.

# Association Page

Reported by M. T. Macfarland, M.D.

## Plan Submitted by Pension Committee for Retirement Income for the Manitoba Medical Profession

M. S. Hollenberg, M.A., B.Sc., M.D., L.M.C.C.

Mr. Harris, our Ex-Finance Minister, in his last budget has given income tax exemptions to funds paid into the registered retirement savings plans.

In his plan he provides that for 1957 and subsequently we will be allowed to deduct in computing our taxable income 10% of our earned income with a limit of $2,500.00 each tax year.

Contributions under the plan must be made in trust to a suitable corporation and applied for the purpose of providing an annuity.

It has also been decided that providing a savings plan is **"registered"** by the end of the taxation year all deposits made in that taxation year will be eligible for tax relief.

The following is a summary of the main conditions of the plan:

(1) The annuity must commence no later than age 71.
(2) The annuity may be payable to a man and his wife on a "last survivor" basis.
(3) It may include a guarantee of not more than 15 years' payments.
(4) No cash option is available at maturity.
(5) Savings plans are not assignable and no "loan values" are available.
(6) Dividends or interest earnings can not be withdrawn in cash but must be used to increase the annuity.
(7) Savings plans funds may be terminated before pension age and the funds withdrawn. In this event the amount returned will be aggregated with income for the year in which the return is received, and the appropriate rate of income tax charged with a minimum rate of 25 per cent.

In the event of death before pension age the total saved will be subject to income tax at the rate of 15 per cent regardless of the individual's tax bracket. Presumably the whole sum, rather than the value net after income tax, is also subject to succession duty.

No decision has yet been given on the method of registering contracts or of notifying payment of "premiums."

As you are all aware, our Pensions and Insurance Committee have succeeded in protecting the doctor's family with Group Life insurance through Manitoba Medical Service.

We are now completing negotiations for an additional $15,000.00 Group Life insurance at the rate of $6.00 per thousand, so that the $30,000.00 insurance will cost us approximately $180.00 per annum.

It is our hope and plan to increase this Group Life insurance to $60,000.00 on the life of each doctor practicing in Manitoba next year. The net result will be to protect the insured doctor's family for the amount of $60,000.00 at an approximate cost of $360.00 per year.

The next objective of our Insurance and Pensions Committee is to establish an annuity for the doctor himself, the income from which shall be sufficient to meet increased living costs in the future.

Since most of us practising medicine do not reach maximum income levels until middle age, it is not possible for us to set aside sums for retirement purposes over a prolonged period. The problems of providing for retirement income are further accentuated because of the increased standard of living required when a doctor's practice becomes established. It is therefore urgent to take every advantage to employ savings and create a fund which will provide realistic income when professional income declines or is cut off completely. Because of the short period during which such savings can be directed and because of the erosion of the purchasing power of the dollar due to inflation, I am presenting the following plan for doctors' pensions.

During the past ten years the rate of inflation in Canada has ranged from 3% to 4.2% per annum. In other words, to keep the purchasing power of the dollar even with the cost of living it must be invested at about 4%. If a real interest return is to be obtained the yield must exceed 4% by a fair margin.

The average return on high grade Canada bonds for the past ten years has been about 3½%. The average return on high grade income producing common stocks (e.g. Bell Telephone) has been about 6.0%. The average return on a well selected, diversified portfolio of common stocks has been considerably more as will be illustrated.

I wish to propose a pension plan using only common stocks.

**Proposal**

I suggest the simplest and most effective way to meet the objectives of the Manitoba Medical Profession and to obtain the benefits necessary is as follows:

1. File for registration with the Federal authorities a fully trusteed pension plan on a money purchase benefit basis.

2. Appoint a corporate trustee to act as custodian of the fund, but with no investment powers.

3. Elect from within the membership a "Pension Committee" authorized to direct investment of the fund with minimum restrictions. Investment powers should permit participation in equities with no specific limitations.

Isolette infant incubator by Air-Shields (Canada) Ltd., Toronto

4. Administer the plan through the established facilities of Manitoba Medical Service.

5. Engage actuarial advisors and counsellors through firms specializing in that field.

6. Appoint auditors on an annual basis.

7. Name competent and experienced investment counsellors.

As the following charts illustrate, representative Canadian common stocks have provided attractive, effective yields over the past ten years. History proves that this record, with some modifications, would extend considerably further into the past. These charts illustrate what could have been done and while it is difficult to forecast the future, it seems logical to look into the past in order to determine what might be expected.

Respectfully submitted,
Michael S. Hollenberg.

**Chart I**

| | *Price Dec. 31/46 | *Price Dec. 31/56 | Total Dividends & Rights 1946-56 | *Total % Gain | Annual Yield % |
|---|---|---|---|---|---|
| Aluminum Ltd. | $ 6.90 | 38¼ | $ 6.90 | 554.3% | 55.43 |
| Bell Telephone | 46.75 | 45.625 | 29.29 | 260.7% | 6.07 |
| B. A. Oil | 13.00 | 46.50 | 6.65 | 308.8% | 30.88 |
| Bank of Montreal | 25.75 | 51.75 | 16.05 | 163.3% | 16.33 |
| Canadian Pacific Rly. | 13.75 | 32.50 | 15.50 | 249.1% | 24.91 |
| Consolidated Paper | 9.875 | 39.00 | 11.56 | 412.0% | 41.20 |
| International Nickel | 36.50 | 101.25 | 27.55 | 252.9% | 25.29 |
| Shawinigan | 25.75 | 83.50 | 15.81 | 285.7% | 28.57 |
| Steel Co. of Canada | 16.20 | 71.00 | 13.84 | 423.7% | 42.37 |
| Moore Corp. | 17.50 | 49.00 | 11.64 | 246.5% | 24.65 |

| | |
|---|---|
| Average Annual Yield | 23.66% |
| Less Projected Rate of Inflation | 4.00 |
| Net Effective Yield | 19.66 |

*Adjusted for Splits, etc.

JAMES RICHARDSON & SONS,
Research & Statistical Department,
Winnipeg, Manitoba,
May, 1957.

**Chart II**

**Projected Results from $2.500.00 Annual Contribution From Age 45 to Age 70**

(1) **Fixed Income Annuities**

Based on Federal rates with 4% interest for a life annuity, commencing at age 70.

**Approximate Value**

| | |
|---|---|
| Principal contributed | $ 62,500.00 |
| Total interest (4% compounded) | 67,442.00 |
| Total value | $129.942.00 |
| Yield, disregarding income tax | 4.0% |
| Less annual rate of inflation | 4.0% |
| Net yield | NIL |
| Annual Income provided | $ 11,862.50 |

(2) **Managed Common Stock Investment**

Based on yield of 1/3 of illustration and using present rates on the lump sum purchase of annuity for life, commencing at age 70.

**Approximate Value**

| | |
|---|---|
| Sum contributed | $ 62,500.00 |
| Total interest (8% compounded) | 134,885.00 |
| Total value | $197,385.00 |
| Yield, disregarding income tax | 8.0% |
| Less annual rate of inflation | 4.0% |
| Net Yield | 4.0% |
| Annual Income Provided | $ 20,280.00 |

(3) **Managed Common Stock Investment**

Based on yield of illustration and using present rates on the lump sum purchase of annuity for life, commencing at age 70.

**Approximate Value**

| | |
|---|---|
| Principal contributed | $ 62,500.00 |
| Total interest and appreciation | 387,794.00 |
| Total value | $450,294.00 |
| Yield, disregarding income tax | 23.00% |
| Less annual rate of inflation | 4.00% |
| Net Yield | 19.00% |
| Annual Income Provided | $ 58,305.00 |

JAMES RICHARDSON & SONS,
Research & Statistical Department,
May, 1957.

## Northern District Medical Society

A meeting of the Northern District Medical Society was held at Dauphin on Thursday, May 16th, attended by the following:

Doctors M. K. Brandt, R. E. Dicks, H. Little, M. Potoski, L. J. Stephen, B. E. Symchych, Dauphin; T. A. Kinash, Gilbert Plains; M. Tanasichuk, Grandview; W. A. Large, Roblin; J. E. Hudson, Hamiota; A. B. Houston, L. R. Rabson, M. T. Macfarland, Winnipeg.

Following coffee provided through the courtesy of Mr. Schmeidl, Administrator, and Mrs. Paul, Matron (Dauphin General Hospital), a very interesting clinical session with presentation of several cases was held in the hospital.

Three cases of Appendicitis in patients of varying ages; one case of nephrotic syndrome in a child; a case of Lymphosarcoma in a female patient; a case of Pernicious Anaemia in an overweight male patient; a case of Polyarthritis in a young male patient; and a case of acute follicular tonsillitis in a young male patient, were presented by several of the local doctors with discussion by Doctors Houston and Rabson.

Following lunch at the Boulevard Hotel, the session reconvened in the Auditorium of the Health Unit when formal presentations were made on Epilepsy by Dr. A. B. Houston and on Surgery on patients in the older age group by Dr. L. R. Rabson.

Dr. J. E. Hudson, President, and Dr. M. T. Macfarland, Executive Director, discussed Association affairs and the meeting ended in time to see Dr. Hudson off to Hamiota in his private plane and to allow other members of the Winnipeg team to see the local Progressive Conservative candidate, Dr. W. G. Ritchie, greet the Federal and Provincial leaders of the Party.

M. T. M.

## Northwestern District Medical Society

A meeting of the Northwestern District Medical Society was held at the Medical Clinic, Virden, Manitoba, on Wednesday, May 15th.

Present were: Doctors W. A. Large, Roblin, President; J. E. Hudson, Hamiota, President, Manitoba Medical Association; J. D. McMillan, Oak River; M. Scherz, Oak Lake; R. S. Harris, J. R. Monteith, Virden; W. W. Grant, S. Israels, M. T. Macfarland, Winnipeg.

There was a clinical discussion of paediatric problems followed by informal presentations by Doctors W. Grant and S. Israels.

Drs. J. E. Hudson, President, and M. T. Macfarland, Executive Director, discussed activities of the Manitoba Medical Association.

Dinner was served in the Central Hotel and a vote of thanks was tendered to Doctors Harris and Monteith for the hospitality extended.

M. T. M.

## Central District Medical Society

A meeting of the Central District Medical Society was held in the new Portage la Prairie General Hospital at 2.00 p.m. Standard time on Wednesday, May 29th.

Present were: Doctors C. M. Thomas, Chairman, Portage la Prairie; T. W. D. Miller, Secretary, Oakville; G. M. Black, G. H. Hamlin, E. T. F. Kent, J. W. Kettlewell, J. M. Neilson, J. C. Rennie, R. E. Renaud, H. K. Stinson, R. K. Watson, Portage la Prairie; J. H. More, Gladstone; J. L. Jenkins, MacGregor; J. E. Hudson, Hamiota; R. E. Beamish, L. G. Cruickshank, M. T. Macfarland, J. S. McGoey, Winnipeg.

Dr. R. E. Beamish presented a paper on the use of Anti-Coagulants. Dr. James S. McGoey dealt with a variety of surgical problems, and Dr. L. G. Cruickshank spoke on Resuscitation. Each paper was illustrated by slides or X-ray plates.

Reception and dinner were held at the Mayfair Hotel, following which Dr. J. E. Hudson, President and Dr. M. T. Macfarland, Executive Director, discussed the work of the Association during the current year.

## Report of Nominating Committee Manitoba Medical Association For Officers 1957-58

Article 11 of the Constitution and By-laws provides that: "The President, First and Second Vice-Presidents, Honorary Secretary, and Honorary Treasurer, and the additional members of the Executive Committee, unless otherwise provided in this constitution, shall be elected at the business session of each Annual Meeting. They shall be elected from nominations, one or more names for each office, to be submitted by the Nominating Committee to the Executive Committee and published in the Association Bulletin at least one month before the Annual Meeting, and from such other nominations as may be made from the floor at the business session of the Annual Meeting.

"The voting shall be by ballot. The poll shall be open during the Annual Meeting for such period after nominations are closed, as shall be decided by the President."

In accordance with the above the Nominating Committee is pleased to present the following report:

President:
Dr. C. B. Schoemperlen, Winnipeg

First Vice-President:
Dr. Edward Johnson, Selkirk

Second Vice-President:
Dr. F. G. Allison, Winnipeg
Dr. L. A. Sigurdson, Winnipeg

Honorary Secretary:
Dr. H. E. Bowles, Winnipeg
Dr. E. Stephenson, Winnipeg

Honorary Treasurer:
Dr. L. R. Rabson, Winnipeg
Dr. A. P. Warkentin, Winnipeg

Member-at-Large:
(Winnipeg) (3 years)
Dr. J. A. Swan
Dr. A. R. Tanner

Member-at-Large:
(Outside Winnipeg) (3 years)
Dr. J. C. Menzies, Morden
Dr. R. F. M. Myers, Brandon.

## Licensed Practical Nurses

Doctor M. R. Elliott, Deputy Minister of Health, for the Province of Manitoba, has forwarded to the Association office a memorandum received from the Registrar-Consultant for Licensed Practical Nurses relative to instructions given by doctors to licensed practical nurses in a private home. The memorandum states in part:

"Periodically I receive calls from Licensed Practical Nurses on private duty in homes stating that they receive inadequate or no instructions from the private physician with regards to the patient's condition and the treatment prescribed or recommended. As the Licensed Practical Nurse legally works under the direction of a private physician in the home, this (1) places her legally in an embarrassing position, (2) does not permit her to give intelligent nursing care to the patient. Therefore it would be appreciated if you could bring this to the attention of the Medical Profession."

An Act to provide for the Training, Examination, Licensing, and Regulation, of Practical Nurses says in part (Section 4, Chapter 204. Revised Statutes of Manitoba, 1954):

(1) If approved by a duly qualified medical practitioner in each case, and subject to subsections (3) and (4), a licensed practical nurse may perform for a patient the duties for which training is provided in an approved school in conformity with the curriculum of studies and program of training prescribed by the council.

(a) in mild types of illness;
(b) in chronic illness of long duration not requiring the services of a registered nurse;
(c) during the convalescence of the patient;
(d) before and after childbirth, where there are no complications neccssitating the services of a registered nurse;
(e) in cases of acute illness;
(f) in any other cases prescribed in the regulations:

and, where required by the duly qualified medical practitioner, the duties shall be performed under the supervision of a registered nurse.

(2) A licensed practical nurse shall not practise in a hospital or other institution, except under the supervision and control of a registered nurse at all times.

(3) A person to whom special licence is issued under subsection (4) of section 8 shall be employed only in a hospital or other institution for the care of the sick or mentally ill, and under the supervision of a registered nurse.

(4) This Act does not, except as herein or in the regulations specifically otherwise provided,

(a) confer on anyone any power, privilege, or right, conferred on a legally qualified medical practitioner under The Medical Act;
(b) confer on anyone any power, privilege, or right, conferred on a registered nurse under The Registered Nurses Act;
(c) confer any authority to undertake the diagnosis, treatment, or cure, of disease, pain, injury, deformity, or other physical disability, by medical, surgical, or any other means, or to practise medicine contrary to The Medical Act;
(d) prohibit the care of the sick
  (i) by members of the family of the patient; or
  (ii) by a domestic servant, housekeeper, nursemaid, salaried companion, or other household helper, whether employed regularly or because of an emergency, if she is employed primarily in a domestic capacity and does not hold herself out, or accept employment, as a person licensed to practise nursing for hire;
(e) prohibit any person from giving aid in a case of emergency;
(f) prohibit the practice of nursing by a person enrolled as a student in a nursing school and preparing herself to be a registered nurse, or by a person approved and enrolled by the council as a student practical nurse and preparing herself to be a practical nurse;
(g) prohibit the practice of nursing in the province by a person who has, in another province or country, a status equivalent to that of a registered nurse or a licensed practical nurse in Manitoba, and whose engagement requires her to accompany and care for a patient temporarily residing in the province during the period of the engagement, if the person having that status does not represent herself, or hold herself out, as being a licensed practical nurse under this Act; or
(h) prohibit the practice of nursing, in any mental institution in the province, by a person who is a student in a course of mental nursing, or a person holding a certificate in mental nursing. S.M. 1953 (2nd Sess.), c.42, s.4.

M. T. M.

## Ringworm Due to Microsporum Canis

During the past year there has occurred in Winnipeg a definite rise in the incidence of ringworm of the scalp and body as a result of infection with microsporum canis. Over seventy individuals with this disease have been seen in the Skin Clinics at the General and Children's Hospital. A minimum period of two months is required for effective treatment during which time the patient, if a school child, is excluded from school. The majority of the patients have been school children.

It has been proven in a number of cases that the human infection was the result of contact with a household pet, usually a cat which was infected with microsporum canis.

In our endeavour to control this infection, we would request your co-operation as follows:

(A) If you see a patient with ringworm of the scalp or body, inquire if such a patient has been in close contact with a cat or dog.

(B) If the patient has been in close contact with such animals, please report the case to the City Health Department (WH 6-0121) giving the name and address of the patient.

The City Health Department will then investigate and arrange for the implicated animal to be picked up and examined by a veterinarian to establish if that animal has ringworm. If it is the wish of the owner of the pet, the owner may take the animal to any veterinarian for examination.

If the examination of the animal demonstrates ringworm, the owner will be advised to: (1) have the animal destroyed, or (2) place the animal in quarantine with a veterinarian until a cure is effected.

Veterinarians practicing in Winnipeg will be advised where hairs or skin scrapings from animals may be sent for microscopic examination or examination by culture.

R. G. Cadham, M.D., D.P.H.,
Medical Health Officer.

## Hobby Show
### 1957 Annual Meeting

Continued success of the Physicians' Art Salon at the Annual Meeting of the Canadian Association and interest created in the Hobby Show of the Manitoba Division, has prompted the appointment of Dr. E. R. Rafuse as Chairman of the Committee for 1957.

Any member of the Association who has a handicraft hobby which would be suitable for showing, is requested to communicate with the Executive Director, 604 Medical Arts Building, prior to September 1st.

M. T. M.

## Southern District Medical Society

A meeting of the Southern District Medical Society was held at 3 p.m. Standard Time on Thursday, May 30th, 1957, in the School Auditorium, Winkler, Manitoba.

Present were: Doctors H. U. Penner, President, C. W. Wiebe, Secretary; B. J. Froese, Winkler; W. M. Colert, A. F. Menzies, J. C. Menzies, Morden, W. E. Artes, Dominion City; E. K. Cunningham, Carman; S. S. Toni, Altona; J. E. Hudson, Hamiota; J. L. Asselstine, M. T. Macfarland, F. R. Tucker, Winnipeg.

Dr. J. L. Asselstine, Child Guidance Clinic of Greater Winnipeg, spoke on the topic "Adolescence Problems." Dr. F. R. Tucker spoke on "Low Back Ache."

Following discussion a delicious turkey dinner was served in the School Library.

At the Business Session new officers were selected as follows:

President: Dr. S. S. Toni, Altona

Secretary: Dr. E. H. Penner, Altona.

Dr. D. G. Irving was named representative to the Executive Committee, M.M.A. for the balance of the year 1956-57 and for the year 1957-58. Dr. C. W. Wiebe was asked to continue as representative to M.M.A. Nominating Committee for the year 1956-57. Dr. W. M. Colert, Morden, was named to the Nominating Committee, M.M.A. for 1957-58.

Dr. J. E. Hudson, President, and Dr. M. T. Macfarland, Executive Director, discussed the work of the M.M.A.

A vote of thanks was moved to the visiting speakers by Dr. Wiebe; also to the local hosts on behalf of the Southern group.

## Victorian Order of Nurses

The Victorian Order has been serving the people of Canada for sixty years. In this Jubilee Year the essential fact which deserves to be widely known is that the Victorian Order nurse will call at the home of any sick person. She works under the direction of the family physician and will visit daily or less frequently depending on the needs of the patient and the physician's instructions.

V.O.N. is a non profit organization. The cost of the nurse's visit is computed yearly. Thus, the cost of each visit in the Greater Winnipeg area and in Transcona is currently $2.25. Many patients can and do pay this fee, others pay only a portion of it, and still others receive free care. A suitable fee in each case is arranged by the nurse following a discussion of circumstances with the family. All such information remains confidential.

For service call WH 2-8529.

## International College of Surgeons Obstetrics and Gynecology Awards

The Division of Obstetrics and Gynecology of the United States Section, International College of Surgeons, announced that two awards will be made for the best manuscripts not exceeding 5,000 words submitted by December 1, 1957. The first prize will be $500 and the second $300.

Contestants must hold the degree of Doctor of Medicine from an accredited college of medicine, and (1) be interns, residents or graduate students in obstetrics and gynecology, or (2) be teachers of obstetrics and gynecology. Fellows of the College are not eligible.

The two successful candidates will be asked to participate in the scientific program of the Division of Obstetrics and Gynecology at the 1958 annual congress of the United States and Canadian Sections, International College of Surgeons.

Details of the contest and the forms in which the manuscript must be submitted may be obtained by writing Dr. Harvey A. Gollin, secretary of the Committee on Prizes, 55 East Washington Street, Chicago 2, Ill.

"The purpose of this contest is to advance the art and science of obstetrics and gynecology, in accord with the principles of the International College of Surgeons and with the aims of the College to extend the frontiers and elevate the standards of all branches of surgery," Dr. Raymond J. Pieri of Syracuse, N.Y., chairman of the Committee on Prizes, said.



## 1957 Annual Convention of National Society for Crippled Children and Adults

Chicago—The 1957 annual convention of the National Society for Crippled Children and Adults —the Easter Seal Society—will be held October 31 to November 2 in Chicago's Palmer House, Dean W. Roberts, M.D., executive director, announced here today.

Prominent authorities who specialize in the rehabilitation of crippled children and adults as well as lay persons interested in non-scientific aspects of the work will participate in the three-day meeting. Speeches, seminars, workshops, clinics and demonstrations will spotlight the newest techniques and latest information in the care, treatment and training of the crippled.

Delegates expected to attend the meeting will come from Easter Seal societies in the 48 states, District of Columbia, Alaska, Hawaii and Puerto Rico.

Dr. James B. Johnson, prominent Newark, Ohio orthopedic surgeon, is chairman of the 1957 convention

## Pan-Pacific Surgical Congress Seventh Congress

The Seventh Congress of the Pan-Pacific Surgical Association will be held in Honolulu, Hawaii, November 14-22, 1957. All members of the profession are cordially invited to attend and are urged to make arrangements as soon as possible if they wish to be assured of adequate facilities.

An outstanding scientific program by leading surgeons with sessions in all divisions of surgery and related fields promises to be of interest to all doctors.

Further information and brochures may be obtained by writing to Dr. F. J. Pinkerton, Director General of the Pan-Pacific Surgical Association, Room 230, Young Building, Honolulu, Hawaii.

## Urology Award

The American Urological Association offers an annual award of $1000 (first prize of $500, second prize $300 and third prize $200) for essays on the result of some clinical or laboratory research in Urology. Competition shall be limited to urologists who have been graduated not more than ten years, and to hospital internes and residents doing research work in Urology.

The first prize essay will appear on the program of the forthcoming meeting of the American Urological Association, to be held at the Roosevelt Hotel, New Orleans, Louisiana, April 28-May 1, 1958.

For full particulars write the Executive Secretary, William P. Didusch, 1120 North Charles Street, Baltimore, Maryland. Essays must be in his hands before December 1, 1957.

# Social News

Reported by K. Borthwick-Leslie, M.D.

Alas, gone are those two months of peace and freedom from the ghosts of gossip and Gordon, haunting the reading of the daily news! To those readers who should be announced, but missed, I apologize. Those ghosts were intermittent in prodding me during the hot weather.

♦

Numerous members have been honored since the last Review. Congratulations to:

Dr. C. B. Schoemperlen elected to membership in the American Bronchoesophagolical Association —a highly restricted organization, and indeed an honor for our Ben.

Dr. O. S. Waugh, in Edmonton at the annual meeting of the C.M.A. was honored, as a senior member of outstanding contribution in medicine, community and country.

A former Winnipegger, Dr. Bertram Feinstein, Man. Graduate, now chief of neurosurgery of Mount Zion Hospital, San Francisco — recently "made" the news as the star surgical performer, opening a new operating room especially equipped for neurosurgery. T.V. and Press Conference were in attendance.

Dr. Emmett Dwyer, regional medical officer for the C.N.R., has been promoted to the rank of Commander Brother of the St. John's Ambulance Association.

Dr. W. Gordon Lamberd, Winnipeg, has been awarded a Fellowship, by the Smith, Kline and French Corporation, for training in psychotherapy and research methods. He has been appointed a Fellow in Psychiatry at the Mayo Clinic.

♦

The National Research Council announced three awards to Manitoba:

1. Dr. Stefan A. Carter, physiology, Mayo Foundation for Medical Research.
2. Dr. Barry Joseph Kaufman, physiology, U. of M. Medical Centre.
3. Dr. Edward Leslie Lansdown, pharmacology and therapeutics, U. of M.

♦

Dr. Alan Klass, Winnipeg, addressed the combined sections of Surgery, Cardiology and Medicine at the annual meeting of the B.M.A. in Newcastle on Tyne, England. Dr. Klass spoke on his new technique of surgical procedure in the sudden obstruction of blood supply to the intestines.

♦

The Winnipeg Clinic announces appointments to its staff: Drs. Robert H. Thorlakson, F.R.C.S. and T. Kenneth Thorlakson, F.R.C.S. in Department of General Surgery.

J. W. Jackson. M.B., Ch.B., F.R.C.S. (Ed.) and V. de C. Magian, L.R.F.P.S., D.L.O. in the Department of Otolaryngology, also J. C. Howarth, M.B., Ch.B., M.R.C.P. (Lond.) in Paediatrics.

♦

Dr. and Mrs. Quentin Jacks, Vancouver, B.C., with the four boys, have been renewing old friendships in Winnipeg. Mighty photogenic, Vann, was that group in your yard.

♦

Dr. and Mrs. Jack McKenty and children have arrived from Montreal to spend a few weeks the guests of proud grandparents Dr. and Mrs. J. M. McKenty, Sr. and Mrs. G. P. White.

Cupid has had a busy summer—among some of his victims are:

Drs. R. C. and Patricia (nee Pickard, daughter of Dr. and Mrs. H. I. Pickard, Oxbow, Sask.) Wightman, have returned from their wedding trip and are located in Park Towers, St. James.

♦

South Burnaby, England, May 18, 1957, Adele Mueller, R.N. and Waldo McLean Yule, B.Sc. '51, M.D. '56 U. of M. were united in marriage.

♦

June 15, 1957, Mary Gretchen Mathers, only daughter of Dr. and Mrs. A. T. Mathers became the bride of W. Shelton, Winnipeg.

♦

July 6, 1957, Marlene Musgrove, younger daughter of Dr. and Mrs. Wilfred M. Musgrove (Bud), became the bride of Dr. Edward Leslie Lansdown, only son of Dr. and Mrs. Leslie Lansdown. This has been a big year for Teddy Lansdown, Honor graduate and scholarship winner this spring — fellowship award and a bride like Marlene! Wow!

♦

June 29, 1957, Frances M. Laidlaw exchanged marriage vows with Morley Carman Sutton, B.Sc., M.D., U. of M. '57. The bride is a W.G.H. graduate of 1956. The young couple have taken up residence in Souris, Man.

♦

July 26, 1957, in Toronto, Ont., Isobel C. Ruthven became the bride of Dr. Thomas Lamont, son of the late Dr. Laurie Lamont and Mrs. Lamont, Winnipeg, formerly of Treherne, Man. Dr. and Mrs. Lamont will reside in Wallaceburg, Ont.

♦

July 27, 1957, Janice Marie Warrington of Weyburn, Sask., became the bride of Donald John Scott, son of Dr. and Mrs. D. L. Scott, Winnipeg, at whose home the wedding reception was held. Mrs. Sidney Larson, Canton, Ohio, aunt of the groom, was one of the out-of-town guests.

♦

June 12, 1957, Marjorie Jane, daughter of Mrs. Massey-Kenway was united in marriage with Dr. James M. Gillies, only son of the late Dr. and Mrs. J. A. K. Gillies. Glasgow, Scotland. Dr. and Mrs. Gillies, after visiting the British Isles and continent, returned to Winnipeg, where Dr. Gillies is with the Cancer Relief and Research Institute.

♦

Engagements announced in the profession are too numerous to mention, but weddings are looming up in near future.

♦

To our new comers, welcome and good fortune:

Dr. and Mrs. Fred Walton, New Westminster, B.C., daughter Jane, August 1, 1957.

♦

Dr. and Mrs. A. M. Homik, a son, Lawrence Alexander, July 16th, brother for Teresa Marie and John Christopher.

♦

Dr. and Mrs. Walter Fox, Lakeland, Kentucky, an adopted daughter, Tannis Lillian, on May 18th.

♦

Dr. and Paul Adams nee Bernier), a son, June 6th, in Langdon, N.D.

♦

Dr. and Mrs. Richard Roe, June 2nd, a son, Stuart Robertson, Tabor, Alta.

♦

Dr. and Mrs. Wm. Fyles, July 18th, a daughter, Gillean Mary.

# Winnipeg Medical Society Committee Reports 1956 - 57

## Report of the Secretary

*To the President and Members of*
*The Winnipeg Medical Society:*

The first meeting of the Council was held May 28th, 1956, then monthly, starting September 7th, 1956. All meetings of the Council were very well attended, with the discussion under the efficient chairmanship of Dr. Stephenson, ranging over a wide field of Medical thought and endeavor but keeping in mind, at all times, the primary aim of this Society.

The Society held nine general meetings. The January meeting was held at St. Boniface Hospital. The February meeting, under the auspices of the General Practioners' Association of Manitoba, proved to be a very successful innovation.

Minutes of all these meetings were recorded and are on file in the offices of the Society.

During the past year this Society lost through death Dr. R. Rawson, Dr. O. C. Trainor, Dr. M. Brookler.

The business of your Society has benefitted from the Constitution and By-laws prepared last year by Dr. David Swartz and no amendments are proposed.

At this time I would like to express my personal thanks to Dr. M. T. Macfarland and his efficient office staff.

Respectfully submitted.

*John A. Swan,*
*Secretary.*

## Benevolent Fund

*To the President and Members of*
*The Winnipeg Medical Society:*

The following is a review of the contributions for the year of 1956-57 of the Winnipeg Medical Society Benevolent Fund.

Contributions of $1,004.00 by 108 members.

There were no disbursements during the year and the members of the Committee were not called together.

Respectfully submitted.

*S. A. Boyd,*
*Chairman.*

## Membership Committee

*To the President and Members of*
*The Winnipeg Medical Society:*

Tendered herewith is a report of the membership committee for the years 1956-57.

The membership in the Society at present totals 465, which may be broken down as follows:

| | |
|---|---|
| Active paid-up members | 331 |
| Active paid-up members (half rate) | 36 |
| Associate paid-up members | 4 |
| Non-Resident paid-up members | 9 |
| Total paid-up members | 380 |
| Life Membership | 26 |
| Free Membership | 22 |
| Non active | 7 |
| Membership fees unpaid | 8 |
| New Members | 22 |
| | 465 |

This represents no increase or decrease over the total membership for 1955-56.

Twenty-two new members have been added to the roll during the year.

Eight members have been lost to the Society during the year, three by death and five have left the province.

Total membership for 1955-56 was 465 and 465 for the current year.

Total paid-up membership for the current year is 380 and 380 for 1955-56, and the number of membership fees unpaid this year is 8 against 35 of last year which is a decrease of 27.

Respectfully submitted.

*James W. Whiteford,*
*Chairman.*

## Community Chest of Greater Winnipeg

*To the President and Members of*
*The Winnipeg Medical Society:*

The following is my report of the comparison of the contributions for 1955 and 1956 of the Winnipeg doctors to the Community Chest. The total coverage for 1956 showed an increase of 40 doctors over 1955.

| | 1955 | 1956 |
|---|---|---|
| Objective | $19,500.00 | $20,000.00 |
| Total Gift | 19,729.00 | 19,330.00 |
| Average Gift | 47.30 | 44.00 |
| Physicians (percentage contributing) | 89.0% | 87.04% |
| Non-givers | 50 | 63 |

In addition, considerable donations which cannot be calculated were given by doctors as a block gift through various staffs and organizations.

This year the campaign was under the chairmanship of K. Roy Pedrick and his group of volunteer business men with the co-operation of the Winnipeg Medical Society.

I wish to thank all members of the Society for their donations to this great work.

Respectfully submitted.

*Robert G. Greer,*
*Chairman.*

## Report of Public Relations Committee

*To the President and Members of*
*The Winnipeg Medical Society:*

A watching brief has been maintained. There have been no significant developments affecting the Winnipeg Medical Society during this term.

Respectfully submitted.

*Murray McLandress,*
*Chairman.*

## Annual Report 1956-1957 of Internists' Section

*To the President and Members of*
*The Winnipeg Medical Society:*

During the period of 1956-1957 this section held three meetings.

On October 16th, 1956, Dr. Maurice Victor from the Massachusetts General Hospital spoke on "Nutritional Diseases of the Nervous System."

On January 14th, 1957, a Business Meeting of the Internists' Section of the Winnipeg Medical Society was held to elect representatives to the "Professional Policy Committee." These representatives were:

Members: Dr. Sydney Israels, Dr. Gerard Allison.

Alternates: Dr. R. T. Ross, Dr. A. R. Birt.

On January 28th, 1957, Dr. H. J. Swan from the Mayo Clinic, Rochester, Minnesota, spoke on "Simultaneous Catheterisation of the Aorta and the Right and Left Sides of the Heart."

At this time an election of officers for 1957 also took place and the following were elected:

President: Dr. C. H. A. Walton.

Secretary: Dr. A. B. Houston.

Two Co-Directors for the Program Committee are:

Dr. T. W. Fyles

Dr. L. Israels

Respectfully submitted.

*E. G. Brownell,*
*Secretary.*

## Report of the Program Committee

*To the President and Members of*
*The Winnipeg Medical Society:*

During the year 1956 and 1957 the following meetings of the Winnipeg Medical Society were held. The titles and speakers were as follows:

Sept. 21, 1956:
"Stone in Poliomyelitis"
Doctors A. J. W. Alcock, J. A. Hildes, J. R. Taylor.
"Atomic Medicine"
Doctor R. J. Walton.

Oct. 12, 1956:
"The Surgery of the Thymus Gland"
Sir Geoffrey Keynes.

Nov. 16, 1956:
"A Review of 100 Cases of Proven Hodgkin's Disease from Winnipeg General Hospital"
Dr. M. H. Campbell.
"A Practical Approach to Mass Casualties"
Lt.-Col. A. C. Derby.

Dec. 14, 1956:
"The Professors' Panel on Rheumatic Heart Disease"
Moderator: Dr. J. D. Adamson.
Members: Dr. L. G. Bell, Dr. H. Medovy.
Dr. R. MacPherson, Dr. J. Lederman.

Jan. 18, 1957:
"The Annual Clinical Meeting," St. Boniface Hospital.

Feb. 15, 1957:
"Problem Cases in General Practice"
The General Practitioners' Association.
Moderator: Dr. D. Hastings.
Members: Dr. F. Henderson, Dr. W. Hart,
Dr. A. Winestock, Dr. R. Flett, Dr. R. Jacques.

Mar. 8, 1957:
"Clinical Uses of Thyroid Hormone Analogues"
Dr. Alastair MacGregor.

April 10, 1957:
"Industrial Psychiatry"
Dr. Donald Ross.

It is a pleasure to thank the members of the Program Committee, the President, and the Executive of the Winnipeg Medical Society for their great help during the year. We wish to express our appreciation to the speakers for their contributions and congratulate them on the calibre of their papers.

Respectfully submitted.

*Robert Cooke,*
*Chairman.*

## Report of Library Representative, 1956-57

*To the President and Members of*
*The Winnipeg Medical Society:*

During the past 11 months 271 Winnipeg Physicians borrowed 1,051 books and 4,323 journals.

There were seven book displays given before meetings of the Society. Evening hours were from 6 - 10 p.m. during the winter, for 61 evenings.

From the Winnipeg Medical Society grant of $1,000.00 to the Library, 58 books were purchased, 62 volumes were bound, and a subscription to Tice's Medicine was paid. $244.00 was paid for student supervision during the evening hours.

Respectfully submitted.

*F. G. Allison,*
*Representative.*

## Public Health

*To the President and Members of*
*The Winnipeg Medical Society:*

As there were no meetings of the Standing Committee on Public Health of the Winnipeg Medical Society there is nothing to report.

Respectfully submitted.

*R. G. Cadham,*
*Chairman.*

## Legislation

*To the President and Members of*
*The Winnipeg Medical Society:*

I should like to report that on 24 January, 1957, a meeting of the Legislative Committee of Fifteen was held to consider a request of the optometrists to legalize the use of the term "Doctor" or its abbreviation "Dr." before their name, and to be allowed to carry out visual training exercises.

Both requests were thoroughly discussed, and the decision was made to oppose both applications. The reasons for the opposition are: (1) a doctorate, and with it the privilege of prefixing the term "Dr." to a name, is a university degree, and should remain so; and (2) the optometrists are inadequately trained to properly supervise and carry visual training exercises, to the detriment of the patient concerned.

The outcome of the above submission is still unknown as nothing final has as yet been settled in the Provincial Legislature.

Respectfully submitted.

*J. H. Crust,*
*Chairman.*

## Representative to Manitoba Medical Review

*To the President and Members of*
*The Winnipeg Medical Society:*

As representative of the Winnipeg Medical Society to the Manitoba Medical Review, I submit herewith the following report:

In the past abstracts of the scientific papers presented at the Winnipeg Medical Society meetings have been published in the Review. This year an effort was made to have speakers publish their papers in the journal in full. Some of these have been published and others have been submitted for publication but in general this has led to less adequate coverage of the society's scientific meetings than in the past.

The suggestion for a Winnipeg Medical Society page in the Review was not implemented during the 1956-1957 season.

Respectfully submitted.

*J. L. Beckstead,*
*Representative.*

## Anaesthesia

*To the President and Members of*
*The Winnipeg Medical Society:*

It is with pleasure that I submit a report on the Activities of the Winnipeg Anaesthetic Society for the 1956-1957 session.

October, 1956 — A dinner meeting was held at the Medical Arts Clubrooms. Dr. Gordon Wyant of the University of Saskatchewan spoke on "The Comparative Anti-siologic Actions of Atropine, Scopolamine and Belladonza in Normal Human Volunteers." At the same meeting Dr. A. B. Dobkin, also from the University of Saskatchewan, spoke on "The Role of the Anaesthetist During A Disaster."

November, 1956 — A general business meeting was held and two films were shown, "Physiology of Anaoxia" and "Therapeutic Uses of Oxygen."

December, 1956 — The Annual Christmas Dinner and Dance was held at Mamma Trossi's.

January, 1957 — Dr. Tass reported on the Annual Meeting of the American Society of Anaesthesiologists which was held in Kansas City in Kansas.

February, 1957 — Dr. Cham spoke on "Cardiac Resuscitation — A Review of the Literature and report of a case that was successfully resuscitated."

Dr. Minuck reviewed a new book, "Clinical Management of Renal Failure," by M. B. Strauss, M.D., and L. G. Raisz, M.D.

March, 1957 — During March the Winnipeg Anaesthetic Society were hosts to the Western Division of the Canadian Anaesthetists' Society for the annual meeting. This was most successful and a large number of out-of-town anaesthetists were in attendance.

Respectfully submitted.

*M. Minuck,*
*Secretary.*


Most doctors feel it is wisest to continue the infant's evaporated milk formula for six months, adjusting it from time to time to meet his changing needs. Evaporated milk processing makes it easier to digest than fresh milk. This is an important point, since digestive upsets and diarrheas are both more difficult to treat and potentially more serious during infancy.

During baby's important first six months, you can count on the *known digestibility* of his individual evaporated milk formula to give him basic growth protection. It is far wiser to give baby this protection than to try to turn him into an adult too early!

**Carnation**

"FROM CONTENTED COWS"

*Optimum prescription-quality in today's trend to the individual formula.*

## General Practitioners

*To the President and Members of*
*The Winnipeg Medical Society:*

Following is a summary of the activities of the General Practitioners' Association of Manitoba for the year 1956-57:

The Executive of the G.P.A.M. meet the third Tuesday in each month.

Two scholarships of $150 each are paid out to two fifth year medical graduates.

The Annual Valentine Dance and Dinner was a huge success this year, with 125 couples attending.

The regular monthly meeting of the Winnipeg Medical Society held on February 15th, 1957, was under the auspices of the G.P.A.M. Chairman of the meeting was Dr. Don Hastings, President of the Association. Members in the panel were: A. G. Henderson, G. Diehl, W. J. Hart, S. Malkin, A. J. Winestock, R. O. Flett, R. A. Jacques and M. J. Ranosky. This meeting was thoroughly enjoyed by all taking part and by all attending. The G.P.A.M. would be pleased to be invited again to participate in another Winnipeg Medical Society Meeting.

A General Practitioners' Day was held at the Children's Hospital on Sunday, February 24th, 1957, under the direction of Dr. H. Medovy, and sponsored by the G.P.A.M. It is hoped that this will become an annual event.

Respectfully submitted.

*S. Malkin,*
*Secretary.*

## Eye, Ear, Nose and Throat

*To the President and Members of*
*The Winnipeg Medical Society:*

In the year 1956-57 up to the present time, this section held three meetings.

Speakers included Dr. H. Medovy, who presented "The Problem of the Partially Seeing Child." Discussion followed regarding the establishment of Sight Saving Classes for School Children. A committee was formed to investigate the above problem.

Dr. W. Alexander presented a paper on "Vertigo and Recruitment" and Dr. J. S. McGillivray summarized the "City Health Department Eye Referrals." Both papers were given in subsequent meetings.

Respectfully submitted.

*Jack A. Rubin,*
*Secretary.*

## Representative to Executive Committee Manitoba Medical Association

*To the President and Members of*
*The Winnipeg Medical Society:*

As representative to the Executive Committee, Manitoba Medical Association, I have the honour of reporting as follows:

I have attended all the meetings of the Executive Committee and have taken part in all discussions when I deemed it necessary.

I have maintained liaison with the committee and I have reported, when necessary, to the Winnipeg Medical Society Executive.

Respectfully submitted.

*Athol Gordon,*
*Representative.*

## Welfare Council

*To the President and Members of*
*The Winnipeg Medical Society:*

A major interest of the Welfare Council of Greater Winnipeg this past twelve months has been an investigation of the psychiatric services of Greater Winnipeg presently available for those citizens unable to afford private care. The Council has surveyed the situation and recommended the following:

1. That a Committee be established to advise for the development of psychiatric services.
2. That a central registry of psychiatric cases be established, and that a psychiatric services centre be established.
3. That a psychiatric consultation and treatment services should be centralized in the "Winnipeg General - Children's Hospital Medical Centre" and organized for adults and children.
4 That follow-up care of discharged psychopathic Hospital and Mental Hospital in-patients should be provided by the Psychopathic Hospital Out-patient Clinic.
5. That follow-up care of discharged St. Boniface Hospital in-patients and psychiatric service to out-patients of St. Boniface Hospital should be given by the St. Boniface Out-patient Clinic.
6. That psychiatric consultation should be an integral part of the University Health Services.

Other problems to receive attention have been a program to study alcoholism in Manitoba, and the establishment of a community centre for the deafened.

Respectfully submitted.

*C. A. Curran,*
*Representative.*

## Radiological Section

*To the President and Members of*
*The Winnipeg Medical Society:*

The Radiological Section of the Winnipeg Medical Society had quite an active year, holding several meetings, one of which was a joint meeting with the Section of Pathology.

Respectfully submitted.

*J. B. Squire,*
*Secretary.*

## Medical History Section

*To the President and Members of*
*The Winnipeg Medical Society:*

This Section held three meetings during 1956-57, at the Medical Arts Club Rooms, each meeting preceded by dinner.

On November 27 the Section heard a scholarly, thoughtful paper by Dr. A. T. Mathers on Philippe Pinel.

On February 13 Dr. H. M. Ross presented an interesting study of Three Medical Men: Pirate, Soldier and Saint. The Pirate was Dover, the Soldier was Beaumont and the Saint was Livingstone.

On April 4 Dr. Howard Reed gave us a very instructive account of The History of Cataract, illustrated by lantern slides. Officers for next year were elected: Dr. Ross Mitchell, Chairman, and Dr. Dwight Parkinson, Secretary. Dr. Mitchell was authorized to represent the Section in connection with the matter of preserving old photographs in the Medical College.

Attendance at the meetings was of the order of a dozen.

Respectfully submitted.

*I. Maclaren Thompson,*
*Chairman.*

## Treasurer

1st May, 1957.

To the Members,
The Winnipeg Medical Society,
Winnipeg, Manitoba.

In accordance with your instructions we have examined the accounts of

THE WINNIPEG MEDICAL SOCIETY
— and —
THE WINNIPEG MEDICAL SOCIETY LIBRARY FUND

and have prepared therefrom and submit herewith the following financial statements for your consideration:

Exhibit A, Balance Sheet, as at 30th April, 1957,
Exhibit B, Statement of Revenue and Expenditure.

We report thereon as follows:

### Balance Sheet

*Cash on Hand and in Bank:*

Cash on hand was verified by actual count as at date of audit. Cash on deposit in General and Library Fund Accounts was verified by correspondence direct with the Toronto-Dominion Bank, Portage and Edmonton Street Branch, Winnipeg.

*Membership Fees Receivable:*

This item represents membership fees unpaid at the fiscal year-end, as shown by the records of the Society, as follows:

| | |
|---|---|
| 1955-56 Fees | $ 42.00 |
| 1956-57 Fees | 82.00 |
| | $124.00 |

The unpaid balances have not been verified by correspondence with the members concerned.

*Government of Canada Bonds:*

The investments of the Society as at 30th April, 1957, were as follows:

| | Cost | Par Value | Approximate Market Value |
|---|---|---|---|
| 3%, due 1966 | $3,031.88 | $3,000.00 | $2,688.75 |
| 3¾%, due 1978 | 1,473.75 | 1,500.00 | 1,443.75 |
| 3¼%, due 1979 | 1,003.75 | 1,000.00 | 895.00 |
| | $5,509.38 | $5,500.00 | $5,027.50 |

These securities, held in a safety deposit box in The Toronto-Dominion Bank, were produced for our examination and in all cases are fully registered in the name of The Winnipeg Medical Society. In accordance with minutes of Council, $1,000.00 par value 3% Government of Canada Bonds, due 1966, were sold during the year.

*Membership Fees Paid in Advance:*

This item represents 1957-58 membership fees received prior to 30th April, 1957.

### Statement of Revenue and Expenditure

The operations of the Society for the fiscal year resulted in net revenue of $1,728.40 on General Fund Account and net expenditure of $952.79 on Library Fund Account. Full details are set forth on Exhibit B attached.

Revenue from membership fees is in accordance with your records, supported by duplicate receipts which were examined by us. All interest has been accounted for on a received basis.

All expenditures for the fiscal year have been approved in minutes of Council, and adequate vouchers were examined by us in support thereof.

* * *

We record with pleasure our appreciation of the courtesies and co-operation extended to us by Council members and staff during the course of our examination. Should any further information or explanations be required in connection with the attached accounts we shall be glad to be of service.

## Auditors' Certificate

We have examined the balance sheets of The Winnipeg Medical Society and The Winnipeg Medical Society Library Fund as at 30th April, 1957, together with the related statements of revenue and expenditure for the fiscal year ended that date, and have obtained all the information and explanations we have required.

In our opinion, the attached balance sheets and statements of revenue and expenditure are properly drawn up so as to exhibit a true and correct view of the state of the affairs of the Society as at 30th April, 1957, and the results of its operations for the fiscal year then ended, according to the best of our information and the explanations given to us and as shown by the books of the Society.

Yours faithfully,
SILL, STREUBER & COMPANY,
*Chartered Accountants.*

**Exhibit A**

### Balance Sheet
### As at 30th April, 1957

| | General Fund | Library Fund |
|---|---|---|
| **ASSETS** | | |
| Cash on Hand and in Bank | $2,555.73 | $1,185.44 |
| Membership Fees Receivable | 124.00 | |
| Government of Canada Bonds—at cost | 5,509.38 | |
| | $8,189.11 | $1,185.44 |
| **LIABILITIES** | | |
| Membership Fees Paid in Advance | $ 82.00 | |
| Surplus: | | |
| Balances as at 1st May, 1956 | 6,378.71 | 2,138.23 |
| Net Revenue Expenditure for fiscal year ended 30th April, 1957 (Exhibit B) | 1,728.40 | 952.79 |
| Balances as at 30th April, 1957 | $8,107.11 | $1,185.44 |
| | $8,189.11 | $1,185.44 |

**Exhibit B**

### Statement of Revenue and Expenditure
### For twelve months ended 30th April, 1957
### General Fund

| | | |
|---|---|---|
| Revenue: | | |
| Membership Fees: | | |
| Active Members | $3,958.00 | |
| Non-residents and Associates | 74.00 | $4,032.00 |
| Bond Interest | | 178.74 |
| | | $4,210.74 |
| Expenditure: | | |
| Audit Fees | $ 75.00 | |
| Bank Charges, including Safety Deposit Box Rental | 6.05 | |
| Catering Expense | 201.40 | |
| General Expense | 51.35 | |
| Sundry Donations | 2.00 | |
| Lantern Slides and Expense | 45.00 | |
| Manitoba Medical Association—Share of office salaries and expense | 1,320.00 | |
| Printing, Postage and Stationery | 720.93 | 2,421.73 |
| Net Operating Revenue for year | | $1,789.01 |
| Deduct: | | |
| Loss on Sale of Government of Canada Bonds | | 60.61 |
| Net Revenue for year (Exhibit A) | | $1,728.40 |

## Library Fund

| | | |
|---|---|---|
| Revenue: | | |
| Bank Interest | | $ 42.37 |
| Expenditure: | | |
| Books and Periodicals Purchased | $ 607.16 | |
| Library Supervision | 388.00 | 995.16 |
| Net Expenditure for year (Exhibit A) | | $ 952.79 |

## Benevolent Fund

1st May, 1957.

To the Members,
The Winnipeg Medical Society Benevolent Fund,
Winnipeg, Manitoba.

We have examined the accounts of the Fund for the year ended 30th April, 1957, and submit the following statement for your consideration:

| | | |
|---|---|---|
| Balance of Fund, 1st May, 1956 | | $4,920.20 |
| Add: | | |
| Income during Year: | | |
| Donations received | $999.00 | |
| Bank Interest | 94.75 | |
| Bond Interest | 45.00 | |
| | | $1,138.75 |
| | | $6,058.95 |
| Deduct: | | |
| Expenses during Year: | | |
| Safety deposit box rental | $ 5.00 | |
| Exchange on cheques deposited | .80 | |
| | | 5.80 |
| Balance of Fund, 30th April, 1957 | | $6,053.15 |
| Represented by: | | |
| Cash on Deposit— | | |
| The Toronto-Dominion Bank | | $4,586.90 |
| Government of Canada Bonds, 3%, due 1966, par value $1,500.00 (market value $1,344.37) fully registered in name of Winnipeg Medical Society Benevolent Fund — at cost | | 1,466.25 |
| | | $6,053.15 |

Donations received during the year are in accordance with duplicate receipts examined by us.

The amount on deposit with The Toronto-Dominion Bank has been confirmed by correspondence direct with the bank.

The securities are held in a safety deposit box at The Toronto-Dominion Bank, and were produced for our examination. All interest has been accounted for on a received basis.

Should any further information or explanations be required in connection with the foregoing statements, we shall be glad to be of service.

Yours faithfully,
SILL, STREUBER & COMPANY,
*Chartered Accountants.*

# Department of Health and Public Welfare

## Comparisons Communicable Diseases — Manitoba (Whites and Indians)

| DISEASES | 1957 | | 1956 | | Total | |
|---|---|---|---|---|---|---|
| | May 19 to June 15,'57 | April 21 to May 18,'57 | May 20 to June 16,'56 | April 22 to May 19,'56 | Jan. 1 to June 15,'57 | Jan. 1 to June 16,'56 |
| Anterior Poliomyelitis | 1 | | | | 5 | 5 |
| Chickenpox | 74 | 81 | 137 | 100 | 598 | 620 |
| Diphtheria | 1 | .... | .... | 1 | 18 | 2 |
| Diarrhoea and Enteritis, under 1 year | 19 | 16 | 12 | 12 | 98 | 70 |
| Diphtheria Carriers | .... | .... | 1 | .... | 9 | 2 |
| Dysentery—Amoebic | .... | .... | .... | .... | .... | .... |
| Dysentery—Bacillary | 1 | 1 | .. | 3 | 4 | 9 |
| Erysipelas | .... | 2 | 1 | 2 | 6 | 11 |
| Encephalitis | .... | .... | 1 | | | 2 |
| Influenza | 13 | 14 | ... | 11 | 69 | 73 |
| Measles | 360 | 442 | 94 | 171 | 2478 | 1012 |
| Measles—German | 13 | 6 | 14 | 22 | 81 | 151 |
| Meningococcal Meningitis | 5 | 2 | | 1 | 10 | 3 |
| Mumps | 29 | 46 | 82 | 94 | 411 | 891 |
| Ophthalmia Neonatorum | .... | .... | .... | .... | .... | |
| Pneumonia, Lobar | .... | .... | .... | .... | .... | .... |
| Puerperal Fever | .... | .... | .... | .... | .... | .... |
| Scarlet Fever | 5 | 7 | 4 | 19 | 67 | 88 |
| Septic Sore Throat | .... | 7 | .... | .... | 7 | 4 |
| Smallpox | .... | .... | | | | |
| Tetanus | .... | .... | 1 | 1 | 1 | 2 |
| Trachoma | .... | .... | .... | .... | .... | .... |
| Tuberculosis | 44 | 47 | 104 | 63 | 209 | 341 |
| Typhoid Fever | .... | 1 | .. .. | .... | 2 | . |
| Typhoid Paratyphoid | .... | .... | .... | .... | | 1 |
| Typhoid Carriers | .... | .... | .... | .... | 1 | |
| Undulant Fever | 1 | . | 1 | 1 | 1 | 8 |
| Whooping Cough | 20 | 5 | 46 | 40 | 103 | 226 |
| Gonorrhoea | 98 | 91 | 86 | 95 | 472 | 612 |
| Syphilis | 9 | 11 | 5 | 5 | 49 | 33 |
| Jaundice, Infectious | 54 | 44 | 10 | 13 | 331 | 168 |
| Psittacosis | .... | .... | .... | 1 | .... | 1 |

### Four-Week Period May 19 to June 15, 1957

| DISEASES (White Cases Only) *Approximate population | *850,000 Manitoba | *880,665 Saskatchewan | *5,404,933 Ontario | *2,982,000 Minnesota |
|---|---|---|---|---|
| Anterior Polio | 1 | .... | 5 | .... |
| Chickenpox | 74 | † | 1965 | † |
| Diarrhoea and Enteritis under 1 yr. | 19 | 1 | .... | † |
| Diphtheria | 1 | — | — | — |
| Diphtheria Carriers | — | — | — | — |
| Dysentery — Amoebic | — | — | — | 1 |
| Dysentery — Bacillary | 2 | 1 | 6 | 2 |
| Encephalitis Epidemica | — | — | 1 | — |
| Erysipelas | — | — | 2 | † |
| Influenza | 13 | 1 | 33 | 2 |
| Jaundice, Infectious | 54 | 76 | 63 | 30 |
| Measles | 360 | † | 1453 | 720 |
| German Measles | 13 | 45 | 299 | † |
| Meningitis Meningococcal | 5 | — | 9 | 3 |
| Mumps | 29 | 11 | 1101 | † |
| Psittacosis | — | — | 1 | 2 |
| Puerperal Fever | — | — | 1 | † |
| Scarlet Fever | — | 2 | 212 | 28 |
| Septic Sore Throat | 5 | 4 | 41 | 21 |
| Smallpox | — | — | — | — |
| Tetanus | — | — | — | — |
| Trachoma | — | 1 | — | — |
| Tuberculosis | 44 | 35 | 98 | 100 |
| Typhoid Fever | — | — | 4 | — |
| Typhoid Para-Typhoid | — | — | — | — |
| Typhoid Carrier | — | — | — | — |
| Undulant Fever | 1 | — | — | 9 |
| Whooping Cough | 20 | 20 | 146 | 1 |
| Gonorrhea | 98 | † | 68 | † |
| Syphilis | 9 | † | 18 | † |

†These figures were not given on their reports.

### DEATHS FROM REPORTABLE DISEASES

June, 1957

**Urban** — Cancer, 58; Diarrhoea & Enteritis, 2; Influenza, 1; Meningitis (meningococcal), 1; Pneumonia, Lobar (490), 3; Pneumonia (other forms), 12; Tuberculosis, 2. Other deaths over one year, 204. Other deaths under one year, 24. Stillbirths, 13. Total, 320.

**Rural** — Cancer, 30; Diarrhoea & Enteritis, 2; Influenza, 1; Jaundice (infectious), 1; Measles, 1; Pneumonia, Lobar (490), 1; Pneumonia (other forms), 7; Tuberculosis, 4; Chickenpox, 1. Other deaths under one year, 11. Other deaths over one year, 189. Stillbirths, 6. Total, 254.

**Indians** — Cancer, 1; Pneumonia (other forms), 2. Other deaths under one year, 2. Other deaths over one year, 5. Total, 10.

♦

**Anterior Poliomyelitis** — The case included here is a late reported one with onset in February.

**Diphtheria** — A sporadic case occurring in an elderly lady from Virden district.

**Undulant Fever** — Laboratory reports have disclosed two more cases too late to be included. Please remember Undulant Fever is a reportable disease. Of the three cases two are veterinary surgeons.

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